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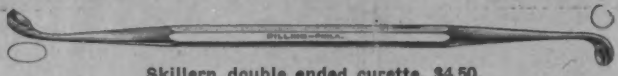

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THE

LARYNGOSCOPE.

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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

SYMPOSIUM ON MAXILLARY SINUS.

SURGICAL ANATOMY OF THE MAXILLARY SINUS.*

DR. HARRY NEIVERT, New York.

In presenting any facts on the anatomy of the maxillary sinus before an audience such as this, I must beg you to pardon me if some facts will bore you. Such a paper must perforce be in the nature of a review; yet even fundamentals may be reviewed with profit. I wish it were possible for each of you to study with me the several hundred specimens which I dissected in the Baugh Institute of Anatomy under the guidance of Prof. J. Parsons Schaeffer.

It is truly fascinating to see how sinuses vary, just as faces do. The shape and size of the face gives us some idea of the size of the sinus. The texture of the skin has often been of aid in determining the thickness of the nasoantral wall, and one can very often tell in advance how easy or how difficult antrum lavage or the other operative procedures will be. A deep canine fossa means a sinus deeply placed; therefore, one must be careful of the curve of the trocar, if a curved instrument is used, so that the point does not come out in front of the antrum into the subcutaneous tissues. Also, a deep canine fossa precludes the possibility of the canine or premolar teeth, if diseased, affecting the sinus.

Pain and tenderness in antrum disease is of course due to direct involvement of the nerve ends in the mucosa. We know that the

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maxillary or second division of the trigeminal nerve receives impulses through the lateral nasal branches of the sphenopalatine nerves as well as through the infraorbital branch, which comes off in the infraorbital canal; and also from the posterior middle and anterior superior alveolar nerves. At times, the infraorbital trunk may be exposed by a dehiscence. It is in the roof or infraorbital wall of the maxillary sinus where dehiscences are most common, because this wall is usually very thin. Pain in antrum disease is frequently felt only in the supraorbital region. The most plausible explanation is that the impulses come through the second division of the third nerve to the Gasserian ganglion and spill over, as it were, to the first or ophthalmic division.

By external palpation, we should learn if the upper deep cervical lymph nodes which lie under the sternomastoid muscle are enlarged. These drain the retropharyngeal nodes, lying in the buccopharyngeal fascia, which receive the lymph from the maxillary sinus. Rarely there will be tenderness deep in the parotid salivary gland, because the subparotid nodes also get some drainage from the nose and pharynx.

Looking into the nose, we recall that the thinnest part of the antral wall in the inferior meatus is the maxillary process of the inferior turbinate. This process occupies the lower part of the V-notch in the hiatus maxillaris, and approximately the posterior and middle third of the upper part of the inferior meatus. This, therefore, is the usual site for entering the sinus, which is about 1 inch posterior to the anterior edge of the inferior turbinate. This also avoids injury to the nasolacrimal opening. At times, this wall may either bulge into the antrum, or into the inferior meatus. If the former, remember that the needle or trocar must be used with caution, especially because in most of these cases we have a deep canine fossa.

The most interesting part of the morphology of the antrum, to me, is the area above the inferior turbinate. The uncinate process, which is part of the ethmoid, as the foundation of this region is of vast importance. Too little attention is paid to it. In 1923, after a large series of experiments, I was forced to the conclusion that its removal should be one of the first procedures in the conservative treatment of chronic antritis, as well as the involvement of the frontal and anterior ethmoid cells. In most cases, the uncinate is intimately related to the bulla ethmoidalis, thereby narrowing the hiatus semilunaris.

Here let us pause to make sure that we are all agreed on the proper nomenclature; hiatus and infundibulum being used by some indiscriminately. The hiatus semilunaris is the opening into the infundibulum ethmoidale. The hiatus is the entrance or door to the dependent recess, the infundibulum, which is of varying depth and breadth, in whose lateral inferior wall is found the ostium maxillaris. The width of the hiatus is determined by the size of the bulla, which is above and lateral, as well as by the plane of the uncinate. The uncinate also determines the breadth of the infundibulum. It may, by curving upward at its inferior end, form a blind pocket, directing exudate from the higher cells into the maxillary sinus. The proximity of the middle concha to the uncinate also contributes to this end. In order to obviate any resistance in this area during irrigation, I make it a practice to shrink this region thoroughly.

The uncinate bridges over the hiatus maxillaris, the latter being the large, irregular opening of the maxillary sinus in the disarticulated skull. It leaves two or more irregular areas about its inferior end, in which no bony skeleton is present. These so-called "undefended areas" are covered by the combined mucoperiosteum of the middle meatus and antrum. It is here that we find accessory ostia and it is quite possible that a cannula passed to the antrum by way of the middle meatus enters through one of them, or is pushed through the attenuated membrane.

Recalling that the maxillary sinus is embryologically an outpouching from the primitive ethmoidal infundibulum and that there is no regular morphology to the definitive nasal structures, we cannot expect to find the ostium maxillaris of definite size or position. The rare true double antrum is due to two primitive outpouchings, whose neighboring walls did not resorb. In my opinion, an analogous explanation accounts for the various septa we find in the antrum, with this exception, that here we must have had one outpouching with several finger-like projections, whose distal contiguous walls did not resorb. A large posterior ethmoid cell may become a surgical maxillary sinus on account of its position in the maxilla, posterior to the true antrum. It still remains a posterior ethmoid cell, because it drains into the superior meatus. How to absolutely recognize such a condition, I don't know.

It is well to recall that the average capacity of the normal antrum is 15 c.c.; that there may be various recesses, zygomatic, alveolar, palatine, infraorbital or prelacrimal. Where the alveolar recess is

marked, the tooth fangs may project and mislead us in interpreting Roentgenograms; therefore, curette carefully in this region. In children, the alveolar process is close to the orbit, thereby making the cephalocaudal diameter small. At about 10-12 years, the floor of the nose and of the antrum are on the same level.

The main blood supply comes from a branch of the sphenopalatine artery by way of the ostium. Additional supply comes from the dental branches of the infraorbital and internal maxillary arteries, which go through the bone to supply the mucosa.

In taking down the inferior meatal wall, be careful about the lower posterior angle. Here we may uncover and lacerate the posterior palatine artery, because at times the posterior palatine canal makes a marked curve forward before reaching the palate. In opening the canine wall, avoid the infraorbital nerve and dental plexus by making as small a hole as is consistent with good surgery.

2178 Broadway.

NONSURGICAL CONSIDERATION OF EMPYEMA OF THE MAXILLARY ANTRUM.*

DR. HARMON SMITH, New York.

Years of endeavor to overcome this condition in the acute and subacute stage without surgical interference probably actuated the Chairman in his request that I should discuss this particular phase of the subject. Years ago, when I first became interested in treatment of antral empyema, neither the means of diagnosis were as accurate as now, nor were the constitutional sequences as well recognized as at the present. Even now, delayed operation and successful treatment must depend upon the co-operation of patient and surgeon, bringing about a gradual amelioration of symptoms by painstaking effort on the part of the surgeon and abiding faith on the part of the sufferer. No miraculous specific is yet at hand to amaze the patient with a wonder-working cure, and all the spectacular procedures of surgery are conspicuous by their absence.

Both the character of the sinus, the type of the infectious organism and the interior nasal conformity unite to either favor or deter the efforts of him who endeavors to cure this malady by treatment. Young patients and those contending with their first involvement yield more readily to treatment than the older patients, and secondary infectious cases cured by operative measures are by no means immune from subsequent involvements. Patients often complain of dry, scabby nares and frequent pharyngitis subsequent to operations. Mucous membranes are never replaced by functioning elements and normal mucus never comes from scar tissue, hence crusts or thick, tenacious mucus invariably follows operations.

Unquestionably, cases where pain and temperature exist, or where treatment has been unavailing, operation is imperative and it is unwise to delay operation for treatment. The frequent puncturing and irrigation of an antrum as a means of treatment is unjustifiable. If the necessity for this procedure arises, it is far better to make a permanent opening into the antrum through the inferior meatus and below the inferior turbinate so that drainage is established and the surgeon can readily irrigate the antrum with a large cannula. Frequent puncturing of the antrum prolongs the treatment — excites

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greater irritation, thickens the lining membrane of the sinus and prevents any natural drainage between treatments. I have seen cases treated for months in this manner, which readily yielded to milder methods when discontinued.

Method of Treatment: Shrinkage of the congested parts adjacent to the sinus ostium with a 1 per cent solution cocain and a 1 per cent solution ephedrin applied on a pledget of cotton in the middle meatus and followed by gentle irrigation with a hot alkalin solution either with the Douglass douche or some of the other well known douches. I employ a powder known as alkanos—using a heaping teaspoon of the powder to a quart of hot water. After this, I pack the nose with strips of cotton soaked in collene or 10 per cent argyrol, which remain *in situ* one-half hour. The patient also sprays the nose every two hours with 1 per cent ephedrin and applies a nose cream, in which there is camphor, menthol and liquid oleo stearate of zinc and returns to the office daily for treatment. In addition, I often give to the patient a hand-bulb suction syringe, which induces a partial vacuum in the sinus and which is again followed by the irrigation. In the office, I employ the Smith's sinus suction syringe attached to the Sorensen pump and induce a 10 degree suction after shrinking the tissues. The syringe not only induces a vacuum but injects into the sinus such medication as it is loaded with. This medication, which is usually some form of colloidal silver, remains in the sinus and lessens the density of the pus and as well stimulates the diseased membrane to healthier activity. When these measures are unsuccessful, I endeavor to irrigate the antrum through the natural opening after cocainization, by means of a small curved blunt-pointed cannula attached to a metal syringe, and in a large majority of cases this effort is successful. Internal administration of one-half gr. of suprarenal extract and one-eighth grain belladonna extract is often indicated, and in cases of pain, hot fomentations and inhalation of steam are most effective. All cases do not yield to treatment, even in the acute stages, and then operative measures must be instituted.

150 East 62nd Street.

THE INTRANASAL TREATMENT OF SUPPURATIVE DISEASE OF THE MAXILLARY SINUS.*

DR. CHARLES J. IMPERATORI, New York.

Unfortunately, in the past, many patients have been treated by the intranasal route, when a radical operation should have been done. The operation that has been generally accepted as the intranasal operation consists in the refractment or pushing away of the anterior end of the inferior turbinate or, in the majority of cases, the resection of the anterior end of the inferior turbinate and the breaking down anteriorly of the lower internal nasal wall of the maxillary sinus. Ventilation and drainage may be thus attained. If the resection of the anterior end of the inferior turbinate can be avoided, it is better, for the lachrimal secretion frequently is diverted, so that patients complain of the watery dropping from the nose. Harmon Smith has called attention to this condition.

It would be better to say that this type of operation is of decided importance and value and should be regularly employed in those cases of maxillary sinus disease that are subacute or of such length of time that the mucosa of the sinus is not particularly diseased. If this is continuously borne in mind, then the accepted intranasal operation will continue to have its usefulness. Attempts to cure chronic maxillary sinus disease by the intranasal route, in the vast majority of cases, ends in failure or prolonged and protracted unnecessary treatment and discomfort, insofar as the patient is concerned. The indications for this operation, in my opinion, should be entirely limited to those type of cases in which the mucosa is not diseased, as shown by Roentgenograms and by the supplementary shadow cast by the instillation of lipiodol within the cavity.

It is impossible to completely remove the mucous membrane of a chronically diseased antrum through the window in the intranasal wall and properly inspect the whole cavity, even though we employ the pharyngoscope, or a similar type of instrument. In those acute cases that do not begin to show improvement within a week following repeated irrigations, and the exudate is in the nature of a flocculent precipitate, and the usual mucus orange-stained clot is not seen, this operation should be employed. Mucous membrane elevations

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of more than a quarter of an inch, as shown by lipiodol injection and continuous flocculent washings, will not yield to the intranasal route, excepting in rare instances. Diseased or devitalized teeth in the neighborhood of the maxillary sinus, as shown by Roentgenograms must of necessity be removed, for they are always potentially the source of recurrent infection. From 10 to 30 per cent of all maxillary sinus infections are due to teeth.

Diagnosis: The suggested method is the following:

First, the posture test, which is obtained by inspecting the nasal passages and observing the presence or absence of pus. If pus is present, remove it, either by suction or with a cotton applicator. Bend the head forward and turn the cheek of the suspected side uppermost. Have the patient remain in this position for a period of five minutes or so. If on inspection of the meatii you again see pus, there is presumptive evidence of maxillary sinus disease. Ordinarily, pus seen underneath the middle turbinate and in the inferior meatus may be considered as coming from the frontal sinus or maxillary sinus. On posterior rhinoscopy following the posture test, usually there will be secretion over the posterior end of the inferior turbinate. The patient may also experience a smelling of foul odor.

Second, transillumination: Voltolini employed transillumination and it was further elaborated upon by Heryng. We should observe on transillumination the following, when the transilluminator is placed in the mouth: *1.* Whether or not there is a bright pupil. *2.* The crescentic tache beneath the orbit. Care should be exercised in distinguishing between the usual crescentic tache and the lachrimal tache. *3.* Subjective light sensation experienced by patient. *4.* Superior transillumination by placing the transilluminator within the orbit and directing the rays downward and observing the palatal transillumination. Logan Turner says the value of transillumination, if employed with proper appreciation of the possible fallacies, assists materially in the diagnosis of maxillary sinus disease.

Third, X-ray: This gives us the depth and breadth and location of the sinus and somewhat of an idea of the mucosa. In order to properly determine the thickness of the mucosa, it is necessary to inject some substance similar to lipiodol.

Fourth, exploration: With a small needle, either by aspiration or by displacement, we determine whether or not there is secretion within the maxillary sinus. The injection of air instead of fluid or suction should not be done. Several fatalities have been reported following this procedure.

The diagnosis is the important thing and, in my estimation, the determination of the condition of the mucosa of the sinus is paramount. In mostly all acute cases it becomes necessary, where the recovery is somewhat prolonged, to enlarge the opening underneath the inferior turbinate so that it is not necessary to puncture each time that an irrigation of the sinus is done. In this instance, where the opening is enlarged and while it is true it is in the nature of an intranasal operation, still I do not feel that these cases should be classified as having been operated by this method.

In my opinion, if the mucosa is not greatly swollen and thrown into large folds, even though under the arbitrary quarter of an inch, too zealous and too repeated irrigations are liable to prolong the infection. We must continuously bear in mind that the mucosa of the sinus is a highly specialized membrane and the cilia of these cells must fuction in order to have the sinus return to normal. Drainage and ventilation should be the important *disidera*. We use our methods of irrigation simply to displace the products of inflammation because this is the easiest and safest method to clear the mucosa of these exudates.

Suction may be applied to remove the secretions from the sinus, but there is a greater possibility of wounding the mucosa by this procedure or aspirating part of the mucosa into the aspirator. The proper placement of a maxillary sinus trocar beneath the inferior turbinate in the soft spot which is located about an inch to an inch and a quarter from the nasal spine and well up beneath the inferior turbinate, or the catheterization of the sinus through the normal opening, can be safely, easily and more or less painlessly done. The resultant material obtained on washing and aspiration should be inspected and its physical properties noted. The examination, microscopically, frequently will assist in determining the chronicity or otherwise of the secretions.

In conclusion, it may be safely said that the cure of suppurating maxillary sinus disease is dependent upon the repair and the refunctioning of the cilia of the mucosa lining the sinus.

17 East 38th Street.

RADICAL OPERATION OF ANTRUM.*

DR. E. ROSS FAULKNER, New York.

The indications for a radical antrum operation seem to vary considerably in the minds of specialists; hence, we find one man considering it the operation of choice in most all diseased conditions, while another would reserve the operation only for cases of polypoid degeneration or bony necrosis.

There is one thing certain, that the conditions which make radical procedure necessary on the sinuses are becoming rarer all the time, for the reason that cases are more frequently treated and cured in the early stages than formerly. Added to this, the more accurate diagnosis of syphilis and its more effective treatment, also the fact that people live under better hygienic conditions than they did in the past, render them less susceptible to various infectious diseases. But perhaps there is one more specific cause, and that is the wholesale removal of infected tonsils and adenoids. While these foci of infection remain in an individual they are constantly subject to recurring attacks of sinusitis, which may result in degenerative changes, necessitating radical operative procedure.

Let us now proceed to discuss the specific indications, which means, what symptoms are manifest in a patient, which make it advisable for him to submit to a short period of discomfort for the prospects of a permanent cure. We will divide symptoms into local and general, and consider the local ones first. These are pain, profuse discharge and frequent acute exacerbations, with both pain and discharge. Pain in an antrum is not usually steady, as it often is in the frontal, and is rarely an indication in itself for radical treatment. The most of such cases are relieved by more conservative operative procedure. Occasionally a complete polypoid degeneration may be accompanied by a feeling of fullness and discomfort, and these cases require radical treatment. Other cases are due to a low-grade osteitis with atrophic changes in the membrane, and while these have been operated on, it is not very usual for them to obtain much benefit. In cases with profuse discharge, the intranasal operation is usually tried first, but if this fails a radical may be necessary, and if all the diseased mucous membrane is removed a good result will follow usually, though in some cases discharge will persist even

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after a radical. The bacteriology of these obstinate suppurative cases might be further studied. I have had two cases of pure culture of staphylococcus aureus in antra which I failed to cure by radical antrum operations. The cases with frequent acute attacks should first be treated by conservative methods, but occasionally will necessitate a more extensive operation. These cases will usually show considerable degeneration of the membrane and some symptoms from general absorption as well.

Let us now consider the general symptoms, and one may state with reasonable certainty that where an antrum is acting as a focus of infection over a prolonged period, the radical operation, or a partial radical, will be the most effective way of dealing with the trouble. These general symptoms may include inflammatory conditions in any part of the body, such as the eye, ear, heart, kidneys, joints or nervous system. I have frequently seen results attend simple drainage in cases where the antrum trouble has been recent, but in most cases the antrum trouble is of long standing and a radical will be necessary. I have seen the simpler measure seem to stir up the focus in these cases and appear to make matters worse. The absorption in many of them comes from a thickened membrane and is accompanied by very little discharge. The pathology which produces various symptoms corresponds to that found in the nasal chambers and other sinuses. Thus, in some cases with pain there may be an osteitis with more or less atrophy of the membrane. The bone in these cases will become so thick as almost to obliterate the cavity. They are difficult to operate on and the results are not brilliant. Another form of osteitis occurs from ossifying granulation tissue and the formation of osteophytes. These cases are rare, but I have seen them in the cadaver. If they are ever diagnosed a radical operation would be indicated. The pathology associated with profuse discharge is not so marked in gross appearance as one would expect. The membrane lining the cavity will be redder than normal and be only slightly thickened, usually in the floor. A partial removal of the membrane only will be indicated. During an acute exacerbation, this membrane becomes greatly swollen, probably analagous to what occurs in a sphenoid. This thin, paper-like membrane can swell up during an acute inflammation to nearly fill the cavity. An X-ray taken during a quiescent period will show very little.

The changes in the membrane in the cases with pronounced absorption symptoms is either a diffuse thickening or thickening with irregular polypoid changes, also areas on the floor or alveolar process may show granulation tissue, with particles of inspissated pus,

yet almost no free discharge. These cases demand complete removal of the lining of the cavity. In old cases of this type, there is sometimes a considerable thickness of very adherent scar tissue, where previous granulations have been or even ossification may have taken place.

Diagnosis of the various conditions which indicate a radical operation are not so difficult as formerly. The symptoms mentioned above should be considered with various other adjuncts. The X-ray is invaluable, but plates should always be taken, if possible, during a quiescent period. Transillumination is also useful, but not so definite as the X-ray. Puncture and lavage: This procedure is very useful to determine whether an opacity in the X-ray is due to retained secretion or thickened membrane. If due to discharge, this will be obvious in the washing. If there is no secretion in the return flow, a thickened membrane is indicated. If the trocar seems to go into a mass of doughy tissue and is very difficult or impossible to wash out, a polypoid degeneration is probably present. A clear return flow from lavage does not give the antrum a clean bill of health by any means, as many an antrum may act as a bad focus of infection and show this phenomenon. The injection of lipiodol to demonstrate the thickness of membrane always seemed to me a superfluous procedure.

Let us now consider the various methods of carrying out the radical operation:

In the Skillern method, which might be called a semiradical, the antrum is entered in front of the inferior turbinate. I have had no experience with this, but the anatomy in some cases would, I think, render this rather unsatisfactory. In people with narrow arches that space is limited and often presents quite a thickness of bone to chisel through. If a large enough opening was made, there would be considerable annoyance from spontaneous drainage from the nostril.

Another radical operation, devised by Denker, is a sort of combination of the Caldwell-Luc and the Skillern operation. It gives excellent access to the diseased region but is more radical than necessary.

The radical operation of choice in practically all cases would seem to be the Caldwell-Luc. This goes through the thinnest part of the bone and gives an excellent opportunity for inspection. I am afraid of appearing presumptious in going into the details of this operation since it is one which most of you have done many times. I sometimes do the initial stage of this operation as an exploratory procedure. A small incision is made and a small area of the canine

fossa exposed. I then wait till I have rendered the field entirely dry. Then chip off the bone about a third of an inch in diameter. Again dry up the field and note the presenting mucous membrane. Incise this, suck out any secretion in the cavity and insert an ear speculum for inspection of the inside. I then put in a pharyngoscope and look over the whole cavity. It sometimes happens that the membrane looks sufficiently healthy to leave it alone. An intranasal opening for drainage is then made, guided by inspection, and the small opening closed with a catgut suture. If I decide to go on with the radical, I enlarge my opening in the soft tissues and the bone and proceed to take out all the diseased membrane. It is important to have a good opening in the soft tissues so that a good exposure of the bone is obtained without strong retraction. Strong retraction injures the soft tissue and gives you a severe, unsightly swelling afterwards. Treat the edge of your wound gently and you will get very little reaction and sew up the soft tissue water-tight with perfect opposition edges when you are through. A good-sized intranasal opening is important and I don't like it made too far forward. Only a portion of the centre of the inferior turbinate should be removed, and leave the anterior end alone. A portion of the membrane of the inferior meatus dissected down and placed inward on the floor of the antrum is a refinement which sounds nice in textbooks but usually curls up when you remove the packing and does not facilitate healing in any way. Packing is easily inserted with a curved director through the intranasal opening. This I usually remove in 24 hours. It should be well covered with vaselin and if removed gently will not cause any bleeding. If bleeding does occur, peroxid will usually stop it and help to cleanse the cavity and break up any blood clot. After-lavage is not done till the third or fourth day and then if there is no discharge the cavity is left alone, as watery solutions tend to produce exuberant granulations. A little plain albolene may be put in with an applicator every day or other day.

Healing process: Knowlton has shown by experiments on dogs that regeneration of membrane does occur in the antrum, even the glandular elements eventually reforming. One finds, years afterwards, considerable filling in of the cavity of the antrum in some cases.

Results: Usually good, occasionally the scar tissue gives some discomfort on weather changes, but if the infraorbital nerve has not been injured this should not amount to much. Persistent discharge is rare but may necessitate a reopening and second curettage of the whole cavity.

101 E. 58th Street.

MAXILLARY SINUSITIS OF ORAL ORIGIN.*

DR. HENRY SAGE DUNNING, New York.

On account of the great developments made during the last few years in oral radiology, and the frequency with which Roentgenograms of the teeth and maxillae are made, maxillary sinusitis of oral origin is found to be a very common disease. For many years, rhinologists have felt that the maxillary sinus was surely their domain, as they have thought in the past that anywhere from 70 to 90 per cent of all maxillary sinus infections were descending infections from the frontals or ethmoids or from the nose itself. I believe that the rhinologists today feel that there are more ascending infections from the mouth involving the antrum than were formerly recognized, but that still the larger percentage of infections of the maxillary sinus come from above downward.

Most of my oral surgery friends feel that the percentage of antral infection of oral and nasal origin is much nearer 50-50. That there are 10 teeth in the upper jaw, two bicuspids and three molars, right and left, and also gingival margin to infect the antrum we, of course, might say that the oral surgeon sees more of these cases originating from the mouth, and that the rhinologist sees more springing from the nasal accessory sinuses. In these days, when so many patients are having their teeth radiographed as a matter of routine, whether they are in trouble or not, latent antral infections are found, and many teeth are removed whose alveoli enter the antrum on gentle curettage. On the removal of many of these teeth, it is found that there is a chronic low-grade infection present in the antrum that has been draining comfortably through the middle meatus for months. This mild infection has taken care of itself very nicely, and the condition has not become acute. We are today giving a great amount of thought to the dental and peridental structure and to the maxillary bones. We are ever on the lookout for bone cysts, neoplasms, impacted teeth, necrosis and infected areas, etc. Few teeth today are extracted by the careful practitioner without being radiographed first. The condition of the bone is known and the bone is often curetted and rongeured, *even though the antrum is opened.* If the antrum is opened, it should be immediately irrigated while there is good anesthesia. for diagnostic purposes. This procedure tells

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the operator at once the condition of the antrum and often saves the patient a subsequent antral washing to learn whether or not the sinus is infected.

I have had many cases during the last year in the hospitals and in private practice in which I have had to make a wound in the maxillae that has caused the mouth to communicate freely with the antrum. In many cases, the antrum has been found to be infected, although there were no symptoms before operation. When the operator has by force of circumstances made a good-sized oral opening into the antrum and has found the antrum diseased, there are two main factors in the subsequent treatment which should appeal to him. These are: *1.* Cure of the antral disease as soon as possible; and, *2.* Closure of the communication between the mouth and antrum at an early date.

If we study the physiologic function of the maxillary sinus, and also its anatomy, we find that the antrum was intended to communicate normally with a comparatively clean cavity, the nose; and that about the same bacteria are found in these two cavities, the nasal and the maxillary sinus. It is evident to the casual observer that Nature never intended the mouth and the antrum to communicate, and never intended the mixing of their different bacteria and secretions. It has been proved clinically that a permanent opening from the mouth into the antrum may produce an ascending infection into the maxillary sinus, later causing a pansinusitis; yet how often do we see the antrum, in these chronic infections of oral origin, irrigated for weeks through the opening in the mouth and packed with gauze? Why should a large oral opening into the antrum be permitted to exist? The mechanics of this treatment is wrong. It is analogous to the housmaid sending her sweepings from the celler through the parlor with its dainty hangings, rather than sending the dust from the parlor, a comparatively clean room, through the cellar, generally not too clean, to the back yard. A gauze drain inserted into the antrum from the mouth often draws up secretions from the mouth by capillary attraction, as does a wick in a lamp, and frequently reinfects the cavity one is trying to cleanse. Up to a few years ago, textbooks were advocating the extraction of a tooth through which to treat a maxillary sinus infection. These books did not stipulate the removal of a diseased tooth that was causing the antral infection, but often a healthy one for drainage purposes only. The sacrifice of a healthy tooth in the old days meant nothing and the establishment of the communication between two cavities containing different secretions and bacteria met with the approval of most surgeons. This method, known as Cowper's, is not advo-

cated now by many authors, although it is often mentioned. The etiology of maxillary infections in the past has not been carefully considered. Not a month ago, a case came to me of maxillary sinusitis, for which someone had just performed a radical antral operation. I was told that there was "a sort of 'gum boil' over the second bicuspid", which was a nonvital tooth. Under procain, I removed the tooth, and the curette entered the maxillary sinus very readily. On exploring the so-called "gum boil" above the tooth, I found it led to necrotic bone. Most of the alveolus of the offending tooth was removed and an area of dead bone as large as a penny was removed from the anterior wall of the antrum just above the alveolar process after a flap of the soft tissues had been turned back. I do not believe that this case was properly diagnosed in the beginning before the first operation. There was some diseased membrane over the end of the infected bicuspid, and I am quite sure that the antral infection would not have cleared up in many months, if ever, without some surgical treatment having been given to the infection of the alveolar process.

This type of infection should be diagnosed early, before treatment is begun, and this cannot be done accurately without taking oral Roentgenograms in conjunction with the anteroposterior plates showing the sinuses. If the infection is a descending one from the frontal or ethmoid sinuses, the case naturally should be treated by the rhinologist, and in some cases by the oral surgeon, if he is familiar with the above sinuses. If the infection is an ascending affair from the maxillary bones and teeth, it cannot be cured alone by the rhinologist unless he is familiar with the mouth and dental structures. In either of the foregoing cases, the cause of the infection must be removed; and if the case is a chronic one, the antrum should be cleaned by a gentle curettage and sometimes by removing the lining membrane, and must be irrigated and drained properly. It also must be given good ventilation. In nearly all of these cases the sinus should be irrigated with warm saline through an opening under the inferior turbinate, and if the infection is of oral origin and there is no opening into the mouth, of course the washings naturally return to the nose and are discharged through that cavity. I do not see why the mouth opening in the oral cases should be packed in any case more than a few days, even if the suppuration has not ceased and although the tissues around the oral openings are not in sufficient healthy condition to bear sutures. In many cases it is advisable to close mechanically the oral opening for a time on account of the suppuration. In these cases the mouth and the maxillary sinus should be separated by a well-fitting "saddle plate"

that does not press at all on the edges of the wound leading into the antrum. No plug or projection that enters the opening should ever be used. I cannot condemn too severely the use of any appliance that tends to establish a permanent opening between these two cavities. This saddle plate is best made of rubber, and covers over the labial and palatal surfaces of the alveolar process, and is generally held by clasps. Of course, this is a temporary affair, and is to be worn only until the suppuration has subsided and the plastic repair of the opening can be accomplished. This plate in a way acts as the valves of the heart, that is, in one direction. It allows of drainage from the antrum downward with gravity under the plate into the mouth, but prevents the seepage upward of the oral secretions into the maxillary sinus. These plates, if constructed properly, never block the drainage and keep the antrum cleaner than any gauze drain can possibly keep it.

In searching the literature, we find little mention of any operative procedure for closing off an opening from the mouth into the maxillary sinus. Little attention has been given to the fact that the mouth and the antrum are two very unlike cavities, that their secretions and their bacteria contents vary greatly, and that they are much better neighbors when they are separated by a barrier of healthy tissues. In cases in which the opening persists between the two cavities, the only permanent method of closing the communication is by means of a plastic operation. A method will be shown by slides later. Infections of the maxillary sinus are often not diagnosed by the dental surgeon and are frequently poorly treated by him. After all that has been written on this subject, it is pitiable to pick up a modern dental journal and to read in a recent number an article advocating the use of the rubber plug, and further to read that "the plug in the antral opening helps to hold the plate in place".

I believe that the rhinologist and the dental surgeon should work more closely together in the treatment of maxillary sinusitis. I feel that it would be better in most cases for the dentist when he discovers an infection of the maxillary sinus to turn the case over to the rhinologist unless he is especially equipped to handle these cases. I have seen many cases treated by dental friends through the mouth, when the infection was a descending affair from the nose, ethmoid and frontal sinus. In many of these cases they accomplished nothing, for the seat of the infection was far removed from their field of activity. *On the other hand, I do think that the rhinologist* should know in every case of antral infection, the conditions of the teeth and the oral tissues. In many cases, he fails to recognize the oral

pathology until after the case has not responded to his treatment. In some cases, after weeks of almost daily irrigations, etc.

Dental radiographs should be taken in all cases and the question of any nonvital teeth that may communicate with the antrum settled before a diagnosis is made. If a tooth looks suspicious in a dental film, its vitality should be ascertained by the usual thermal test, ethyl chlorid spray, electric current or some other means. If the tooth is found to be nonvital, even though it has never been filled, it takes considerable courage on the part of the operator at times to convince the patient that it should be removed. In these cases, I nearly always obtain the consent quite readily to make an exploratory incision over the roots in question, telling the patient that I will not extract it unless it is found to be infected and that it is draining into the floor of the antrum. This generally appeals to the patient as being fair and in this manner I often save for them very valuable teeth that are innocent, even though the dental X-rays may lead one to think differently. Sometimes the radiograph does not show any infection at the root of a tooth, for the reason that the tooth has recently become nonvital and there has not been time for a granuloma to form or any bony changes to take place that would show in the film. At other times, the X-ray shows shadows cast by double septa on the antral floor. These teeth are, nevertheless, the cause and must be removed. My method in these cases, after finding such a tooth to be infected, is to remove it at once and then to irrigate the antrum through the exploratory incision. If the return flow shows pus, I then immediately enlarge my incision, extending it anteriorly to the canine fossa and posteriorly to the last molar. I then lay back the entire labial fold of soft tissues and expose thoroughly the anterior of the antrum. By means of chisels, curettes and the rongeur, a large opening is made into the antrum, through which a careful inspection of the antrum can be made. Incidentally, the end of the tooth socket can be seen and carefully curetted. If there are polypoids present, they are gently removed and also any necrotic lining membrane removed. Great care is taken to remove all diseased tissue without injuring unduly any of the healthy lining membrane. The cavity is then carefully wiped out two or three times a week with a piece of gauze and then irrigated. The nose has been cocainized previously after the antrum was found to be infected and then a fair-sized opening is made under the inferior turbinate by means of a punch and a curette. A large piece of iodoform gauze is then inserted through this opening and is pulled into the antrum by a tenaculum inserted through the oral opening in the antrum. Pulling a fair-sized piece of gauze about one-half-

inch wide, doubled generally, into the antrum toward the oral opening already made, draws the torn edges of the nasal mucous membrane into the antrum or at least invaginates it sufficiently to cover, in most cases, the rough edges of the nasal opening. This, of course, has two advantages. It covers the bony margins of the opening, thus causing almost primary healing over the edges of the bony wound in the lateral wall of the nose and this also prevents the opening from closing, as the bony edges are now covered over by mucous membrane and granulation of this wound is prevented. Enough gauze is pulled into the antrum to fill the cavity comfortably, but none of the gauze is allowed to enter the mouth wound. This generally prevents subsequent hemorrhage and, I think, stimulates granulations in the antrum. The oral opening is then closed carefully by a plastic operation of some kind, including the tooth socket. If the antral infection has been very severe, a second piece of this gauze may be inserted between the flaps to give better drainage. This piece is removed the next day. The nasal piece is allowed to remain for 48 hours and then is gradually removed by degrees, sometimes not entirely for four days. Then after all gauze has been removed, the antrum is irrigated daily with warm normal salt solution and all sutures removed in about three to five days. This operation, I think, can best be performed under local anesthesia, nerve blocking and by swabbing thoroughly the inside of the antrum with cocain, about 5 to 10 per cent, care being taken, of course, not to use too much. The patient is also given morphin, as a rule, before operation. I do most of these operations in the office and, if necessary, have a nurse go home with the patient and remain with them the first night. Most of these cases clean up in about 10 days to two weeks, even though they have been very chronic.

This operation has three distinct advantages, I believe: First, the oral pathology is removed; second, maxillary sinus pathology is obliterated; third, good nasal drainage is established.

The above three objectives are gained by one operation at one time and eliminates separate operative procedures to correct the nasal and oral conditions.

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DIFFERENT TYPES OF DEAFNESS — A STUDY OF THE ETIOLOGY, METHODS OF DIAGNOSIS, PROPHYLAXIS AND TREATMENT.*

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Any discussion of the subject of deafness must of necessity be incomplete. Our knowledge of the pathologic changes in the auditory mechanism that are directly responsible for impairment of hearing is so incomplete that it is not yet possible to place deafness on a definite clinico-pathologic basis. Until such a relationship can be established one is forced to rely largely upon clinical signs and symptoms to determine the site of the lesion and the method of treatment to be followed.

While it is not possible in every instance to determine the exact location of the lesion, or to state positively that a lesion in some particular part of the auditory system will not extend to involve some other portion, there are, nevertheless, certain characteristics which warrant the classification of cases at the time of examination as perceptive, conductive, progressive or otosclerotic types.

It would seem advisable to give a brief definition of these terms and summarize the findings characteristic of each type of deafness before proceeding to a discussion of the etiology, diagnosis and treatment.

1. *Perceptive Deafness:* This term presupposes a lesion somewhere in the perceptive mechanism, either along the pathway from the middle ear, in the cochlea or acoustic nerve, or in the brain center itself. The patient complains of deafness, vertigo and tinnitus, one or all of which may be present. The functional tests in this type of case reveal unilateral impairment more often than bilateral. Conversational voice and the low and medium forks are heard normally in early cases. The higher forks, those above 3,000 double vibrations, and the Galton whistle are either not heard at all, or the length of time for which they are heard is markedly diminished. The Weber lateralizes to the unaffected ear, or to the better ear if both are involved; Rinné is positive, and the Schwabach is shortened. Hearing of low and medium tones by air conduction is normal or only slightly reduced, while by bone conduction there is a

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very definite shortening. Testing with the audiometer shows a depression in the higher tonal range. Examination shows in most instances a drum membrane of normal appearance and no evidence of disease in the Eustachian tube.

2. Conductive Deafness: This type of deafness is the result of interference with the transmission of sound waves to the internal ear. The lesion may be confined to the external or middle ear, or both may be involved. The onset is often sudden. Cases of long-standing differ from the acute only in degree of impairment. Examination of acute cases reveals acute inflammation or congestion in the middle ear, or acute obstruction of the external canal, while in chronic cases one finds changes varying in degree and extent. The functional tests reveal impairment of hearing for spoken and whispered voice and for the lower forks. Weber lateralizes to the worst ear. Schwabach is normal or increased. Air conduction is markedly diminished. Rinné is negative in most cases but may remain positive if the obstructive process is not too advanced. The audiogram is a curve with the diagonal down in the low tones.

3. Progressive Deafness: Progressive deafness, in this paper, is to be understood to refer to those cases who give a history of, or in whom repeated tests have shown, a progressively increasing deafness, and in whom the functional tests show a more or less marked degree of impairment for all tones of the musical scale. In the early stages the loss is usually more marked for either the high or the low notes, but as the disease progresses, impairment is extended to include the whole range of tones. Remissions and exacerbations are not unusual. The former may be spontaneous or may result from treatment of the middle ear or from improvement in general physical condition; the latter are induced by acute infections, fatigue, nervous shocks, mental anxiety and so on.

Examination of patients with progressive deafness always shows evidence of chronic middle ear disease. Functional tests vary with degree of impairment. Diminished air conduction is a constant finding, while bone conduction is normal or reduced in the majority of cases. In the greater percentage of these cases the impairment is bilateral, but one ear may be affected more than its fellow.

4. Otosclerosis: This term is employed to designate those cases of impaired hearing which develop as a result of changes in the labyrinthine capsule, independent of the health or disease of the middle ear. If the disease is located at the oval window, producing fixation of the stapes, the symptoms are identical with those due to other pathologic processes that cause rigidity of the conductive mechanism. If, however, functional tests give the Bezold triad, and exam-

ination shows the drum membrane normal and tube patent, thus excluding tubotympanic disease, one can safely make a diagnosis of primary stapedial fixation. If, however, the disease in the capsule is active at a distance from the oval window, the resulting symptoms are those typical of nerve deafness, and differential diagnosis becomes more difficult. Cases of otosclerosis that begin with symptoms of nerve deafness eventually develop the characteristic signs of stapes fixation. That otosclerosis may develop in patients with evidence of tubotympanic disease cannot be denied, but the occurrence of the two conditions in the same ear is no evidence that either lesion is dependent upon, or influenced by, the other.

Analysis of Cases: In order to determine the relative frequency of the different types of deafness and the constancy of results obtained by the tuning forks and audiometer in each type, I have made a brief analysis of the records of the last 163 consecutive patients with impaired hearing examined in the private practice of Dr. George M. Coates. Among this number I found 109 cases of progressive deafness, 38 of perceptive, 16 of conductive, and none of otosclerosis.

In the progressive cases, the Rinné test was positive in both ears in 39 cases, negative in both in 30 cases, positive in one ear and negative in the other in 40 cases. The low fork (50 d.v.) and high fork (4,096 d.v.) were both heard bilaterally in 17 cases, and neither was heard in either ear in 24. The low fork was heard in both ears in 35 cases and was absent in both in 45, while the high fork was retained in both ears in 40 cases and lost bilaterally in 39. Bone conduction showed an actual increase in the right ear seven times, in the left, 11, and in both, four; it was normal or decreased on the right in 102 cases and on the left in 96. The audiogram tended to one of three curves, *viz.:* a straight line, a curve with the diagonal down in the low notes, or a curve with the diagonal down in the high notes. Among these cases there were 22 in whom there was a history of deafness in the family.

The cases of perceptive deafness showed a bilateral positive Rinné 31 times and bilateral negative only once. Low and high forks were both heard bilaterally in nine cases. Low fork heard bilaterally in thirty-three cases, the only instance in which it was absent being five cases of total unilateral deafness. Bone conduction was normal or decreased in 36 of the 38 cases, the greater number showing a definite decrease. The audiogram was a curve with the diagonal down in the higher notes.

Of 16 cases of conductive deafness, eight gave a bilateral negative Rinné and three a bilateral positive. Hearing for the low fork was lost bilaterally in nine cases and retained bilaterally in four. The

high fork was heard bilaterally in all. Bone conduction was actually increased twice bilaterally and once unilaterally. The curve of the audiogram showed the loss in the lower tones.

ETIOLOGY.

Because of the importance of this phase of the subject, I shall discuss somewhat in detail the etiologic factors that may be responsible for each type of deafness.

The sources of origin of perceptive deafness may be conveniently considered under the following headings:

1. Toxins: It is only in comparatively recent years, since toxins have come to be recognized as exerting such profound influence on general health, that their significance as a cause of deafness has been accepted. The role of acute infectious diseases in the production of toxins is one of extreme importance, and because these diseases are usually encountered in early childhood, the profound degree of deafness which they may produce is not generally appreciated. The large number of these cases that become deaf with no evidence of having had any disease of the middle ear forces one to the conclusion that the deafness is not due to middle ear pathology, but must be the result of some poison which affected the perceptive mechanism. Furthermore, our knowledge of the middle ear prevents the belief that deafness severe enough to be a handicap can be due to disease of this part of the auditory mechanism alone. The problem therefore resolves itself into some injury to the auditory nerve by toxic products disseminated during the course of an acute disease, or over a longer period from some focus for which the acute disease was responsible.

The most extensive study that has been made of deafness resulting from acute infectious diseases is that by Shambaugh[1] and his assistants. Their study included more than 3,000 deaf children, of which number 1,192 were cases of acquired deafness. It is interesting that in 87 per cent of these cases the loss of hearing developed before the age of 10 years, the period during which most children acquire the acute infectious diseases. Practically all of the acute infectious diseases were represented in this series, but in only a very small percentage of the cases was any evidence or history of there having been disease of the middle ear, so that we are justified in assuming that the ear disturbance was the result of a toxic neuritis.

In mumps, which is a more frequent cause of deafness than is generally appreciated, there is very rarely any involvement of the middle ear. When the tympanum is affected, the labyrinth is also not infrequently invaded, in which case there may be complete loss of both cochlear and vestibular functions. The observation that

vestibular symptoms are rare and the deafness profound and permanent leads to the conclusion that the process in most instances is neuritic in character, a conclusion that is strengthened by the cases studied by Willcut[2].

Deafness resulting from focal infections is encountered more frequently among adults than among children. The importance of adenoids as a cause of deafness has long been recognized, but it remained for Emerson[3] to stress the fact that adenoid tissue, as it atrophies with age, may harbor in its depths an infection that may from time to time show acute exacerbations, with the production of toxins that may affect the eighth nerve. While adenoids and tonsils are the most frequent sites of focal disease one must recognize the fact that often after complete removal of these tissues there is hypertrophy of adjacent lymphoid tissue that may harbor infection. Abscessed teeth and peridental infections are less frequent but none the less significant foci. Although the exact relationship between ear disease and paranasal sinus infection is not definitely established, the frequency with which they coexist is evident from Fowler's[4] examination of 100 children with ear disease, in 86 per cent of whom he found evidence of sinus infection. Distant foci, such as infections in the gall bladder, gastrointestinal tract, genitourinary system, and so on, may cause a neuritis of the auditory nerve.

Among the toxic elements drugs have a rather unimportant place, but in seeking for a cause of deafness one must not overlook them. The most frequent offenders are quinin, salicylates, arsenic, alcohol and nicotine. Deafness from drugs is usually only temporary, but may be permanent and total. Mercury may produce auditory paralysis or labyrinthitis with destruction of the cochlear filaments. Dickie[5] mentions a case of acoustic neuritis due to scopolamin-procain anesthesia, and a case of permanent deafness believed to have been caused by radium therapy.

2. *Traumatism* is the etiological factor in a small percentage of cases. Deafness may result from fracture of the temporal bone, which involves the cochlea either by direct fracture or by concussion, or the nerve may be impaired by concussion in the absence of fracture. According to Grove[6], longitudinal fractures of the temporal bone are most frequent and damage the labyrinth by concussion rather than by fracture. Transverse fractures are rare, but when present completely destroy both vestibule and cochlea. Destruction of the labyrinth by bullets and other foreign bodies and crushing by lateral blows accounts for a few cases.

3. *Occupations:* Noise is responsible for the largest number of cases of perceptive deafness that are directly traceable to occupa-

tion. Boilermakers and workers in factories where there is constant noise suffer loss of hearing characteristic of labyrinthine disturbance, due probably to degenerative changes in the auditory nerve filaments. Among 450 boilermakers, Sacher, quoted by Dickie[5], found only 21.6 per cent with normal hearing. Deafness from sudden loud reports, as cannon, and among soldiers on rifle ranges and in action, probably has its origin in labyrinthine hemorrhage or sudden paralysis of the nerve.

Workers in lead are subject to deafness characteristic in that only the acoustic fibres of the eighth nerve are affected. Arsenic, phosphorus and anilin dye workers may also suffer a neuritis of the eighth nerve, but both vestibular and acoustic fibres are affected.

Nerve deafness of caisson workers is most likely due to labyrinthine hemorrhage or embolism as a result of the sudden diminution in atmospheric pressure incident to leaving the caisson. In severe cases the deafness is profound and permanent.

4. Leukemia and Anemia: Deafness in those suffering from leukemic disease may be due to sudden hemorrhage into the labyrinth (Fraser[7]), with complete loss of cochlear and vestibular functions. Apart from hemorrhage, deafness may also be due to degenerative changes in the spiral ganglion cells and nerve endings. In severe anemias, impaired hearing, accompanied by symptoms of vestibular irritation, is occasionally encountered, for which there is no explanation, other than a diminution in the blood supply to the inner ear.

5. Syphilis: As a cause of deafness this disease is not so frequent as has formerly been believed, despite the known vulnerability of the eighth nerve to the syphilitic toxin. Shambaugh[1] found syphilis a probable cause in only 24 of 1,192 cases of acquired deafness. In 500 cases, Hays found only three in which the disease may have been a cause, and in the 53 perceptive cases analyzed by the author, there was no evidence of syphilis in any case. Kerrison[8] recognizes four clinical types of labyrinthine syphilis.

6. Ménière's Disease: The deafness which is one of the cardinal symptoms of Ménière's syndrome is typically perceptive. The conception of this syndrome, based upon the description of Ménière's original case, as an entity due to intralabyrinthine hemorrhage, is a fallacy which is being generally recognized. Such a symptom-complex does not necessarily mean an exudate in the labyrinth. A toxic neuritis, depending upon the severity of the infection, may cause slight impairment or complete loss of function of one or both branches of the eighth nerve and may be due to focal infection in the teeth, tonsils or other parts of the body. This observation is

borne out by the almost immediate relief afforded in some of these cases by removal of some focus.

7. Presbyacousis: The functional tests in senile deafness give results that do not differ from those in other cases of nerve deafness. In addition, these patients show arteriosclerosis and hypertension and a high cholesterin content of the blood. Berberich, quoted by Dickie[5], lays stress upon the presence of an arcus lipoides of the membrana tympani, which he says is always present in senile deafness and is accompanied by an arcus lipoides of the cornea, and increased blood cholesterin. That the hypercholesterinemia may be a causal factor is a theoretical possibility.

8. Congenital Deafness and Deaf Mutism: That the deafness in these two conditions is the result of pathology somewhere in the perceptive mechanism cannot be questioned, because in most cases the impairment is far too great to be due solely to interference with sound conduction. That congenital deafness is the result of a developmental defect in the auditory apparatus is the most tenable theory and is substantiated by certain congenital defects that have been observed in cases of deaf mutism, among which may be mentioned bilateral absence of the external auditory meatus, absence of the modiolus and lamina spiralis, rudimentary cochlea, complete absence of both labyrinths and both auditory nerves. Of etiological factors, heredity and consanguinity, provide the only basis for study. Shambaugh[1] in studying 1,928 cases of congenital deafness, found 723 in whom these two factors apparently played an important part. Cases of deaf mutism are attributable to the same causes but not so frequently as one might suppose, as is evidenced by Kerrison's[9] summary of the statistics of Mygge and Love.

Acquired deaf mutism is the result of a lesion which attacks the auditory mechanism in early life before the individual has acquired or is able to retain the powers of articulate speech and is rarely, if ever, transmitted by marriage. The lesion is unquestionably due to a process which attacks the auditory nerve from within the cranial cavity, meningitis, or to a suppurative labyrinthitis developing from intratympanic suppuration, or to toxins which reach the inner ear by the bloodstream.

The causes of conductive deafness may be devided into two classes, predisposing and exciting.

Among predisposing causes, the most important are interference with normal nasal respiration and normal aeration of the middle ear. The majority of cases of acute otitis in children occur in those suffering from adenoids, and in acute exanthemata the greater number of middle ear complications are found in those children whose naso-

pharynges are occluded by adenoid masses. Children of the poorer classes, usually undernourished, are more frequent sufferers from adenoids and also from middle ear diseases than those in a more fortunate condition.

Tonsils influence deafness when by hypertrophy they obstruct respiration, or when infected act as a constant source of irritation. Adhesive bands in the fossa of Rosenmüller interfere with proper action of the muscles in opening and closing the tube mouth. Hypertrophy of the lateral pharyngeal bands may seriously interfere with tubal ventilation. Marked deviations of the nasal septum, spurs, polypoid or hypertrophied turbinates predispose to deafness by occluding the nasal airways or by their irritative action on adjacent tissues.

Of the exciting causes, the common cold is probably the most frequent. When we consider the mucous membrane of the Eustachian tube as a continuation of that lining the nasopharynx, and realize the enormous congestion which characterizes acute nasopharyngeal infections, we can visualize just what takes place in the tubotympanum and we wonder that we do not see more chronic tubal obstructions. One sees much the same process in the acute infectious diseases, most of which are ushered in with an acute coryza. Infections in the sinuses, particularly those whose drainage is directed posteriorly, may produce middle ear disease and subsequent conductive deafness. The importance of infections in the sphenoid sinus as an etiological factor in the production of conductive deafness was brought out by Sluder[10], who showed the intimate relation of the sphenoidal wall to the Eustachian tube and the ease with which a sphenoiditis can produce irritation in the tube. Vasomotor rhinitis, hay fever and similar conditions, swimming, improper douching and blowing the nose are also factors in the production of impaired hearing.

Just as certain occupations were found responsible for perceptive deafness, so also are some the cause of conductive impairment. Workers in dust-laden air, railway employes, auto racing drivers, bakers, janitors and granite workers are subject to pharyngeal irritations and therefore to tubotympanic disease, as are also bookkeepers, stenographers and other office workers, who are subjected to the superheated air of the office all day and then the sudden change to cold outdoor atmosphere. Irritative gases and fumes, phosphorus, arsenic and mercury are responsible for some cases.

Foreign bodies in the external canal and atresia of the canal from exostoses or disease of the soft tissues are not uncommon causes of deafness of the conductive type.

The etiology of progressive deafness, as that term is used in this paper, is a combination of the factors responsible for perceptive and conductive deafness. Improvement may occur with removal of one or more of the responsible factors, or the disease may progress only when there is an acute exacerbation of infection or during intercurrent disease. Emerson[11] believes that all cases of progressive deafness are due to infection.

As yet we have no definite knowledge regarding the etiology of otosclerosis. Practically all authors agree that the disease is more frequent in the female. Heredity is a factor in approximately 50 percent of the cases. Fraser[12] reports that Hammerschlag traced otosclerosis through two, three and even four generations of the same family. The association of otosclerosis with blue sclerotics and osteogenesis imperfecta which show a strong hereditary tendency has long been recognized.

Bryant[13] believes the etiology rests upon focal infection, endocrine dysfunction and heredity. Drury[14] thinks otosclerosis is dependent, not upon the disturbed function of any single member of the endocrine system, but rather is the result of a metabolic disturbance induced by endocrine dysfunction. Kopetzky[15] found a disturbance of calcium metabolism in 100 per cent and increased blood uric acid in 76 per cent of 67 cases.

The influence of pregnancy on otosclerosis is recognized to such an extent that the artificial interruption of pregnancy has been suggested by some as a prophylactic measure. Some observers have found that the onset can be traced to the first pregnancy, and that after parturition there is improvement for a time, the symptoms becoming more pronounced at the onset of another pregnancy or at the climacteric. The effect of emotional states on the progress of the disease has also been recognized. Otosclerotic soldiers became much worse during the war. Sudden fright, profound grief and severe mental shocks likewise have a deleterious effect.

Cahill[16] presented the theory that the new bone formation in a typical case of otosclerosis is a true benign tumor developing about congenital foci.

Syphilis was formerly considered an important factor in the causation of otosclerosis. Haberman held that this disease was the direct cause. Denker studied 27 cases in which the clinical histories were carefully recorded during life and in which the tissues were examined microscopically, postmortem, and concluded that there was no evidence that syphilis was ever directly responsible. Association of the two diseases is occasionally observed.

The similarity of the bony changes in otosclerosis to those in rickets, osteoporosis, Pagets' disease and other bone diseases has led to the theory that diet may be a factor, and especially that vitamins may play an important part. With this in mind, Barlow[17] studied the effect on the capsule of rats, of a diet deficient in Vitamins A and D, and found no pathologic capsular changes as a result of the deficiency.

DIAGNOSIS.

To make an accurate diagnosis of the type of deafness, to determine the site of the lesion, and to search out the etiological factor, requires time, patience, meticulous attention to detail and painstaking thoroughness in the examination. Because of our limited knowledge of underlying pathological changes, a clinical diagnosis is the only satisfactory one at present, but we may amplify this clinical diagnosis by correctly interpreting various self-evident pathological data, such as abnormal conditions in the nose and throat, the mobility of the drum membrane and ossicles, and the patency of the Eustachian tube. Further data may be added by a careful history and the various tests for hearing.

To be of real value a history must contain data relative to, not only the present condition, but also previous conditions that may have a very definite bearing on the deafness, *viz.*, previous acute infections, gastrointestinal and genitourinary diseases, hyper- and hypotension, abscessed teeth, earache, neuralgia and deafness in other members of the family. The history of the present condition, should contain the following: date and mode of onset; exciting cause; presence or absence of otalgia, tinnitus, vertigo, dizziness, discharge from the ear, or neuralgia of any of the cranial nerves; has deafness been progressive, intermittent or stationary, and is it influenced by atmospheric changes, overwork, anxiety or constitutional factors; occupation and environment; and general health.

Examination: Inspection of the membrana tympani may reveal much or little. Retraction of the membrane, loss of normal lustre, broken cone of light, areas of atrophy or thickening, perforations, scars, discharge, destruction of the membrane and ossicles, any one or more of these may be seen at a glance, and each may be of importance in arriving at a final diagnosis. The presence of a pinkish blush, seen through the membrana tympani, and supposedly due to congestion of the mucous membrane covering the promontory, has long been regarded as more or less pathognomonic of otosclerosis. Josephson[17a], in a large series of cases of progressive deafness, has observed a characteristic and constant injection of vessels of the drum membrane and inner end of the external canal.

By using a Sigel's otoscope one may test the mobility of the drum membrane and ossicles. The membrane may show marked exaggeration or limitation of motion, or certain areas may move more freely than others, or wide excursions of a relaxed membrane may be seen to exert practically no influence on the malleus handle and therefore on the ossicular chain.

To determine the condition within the Eustachian tube, inflation per catheter with compressed air is the method of choice. The Politzer method is much less satisfactory. The sounds heard through the diagnostic tube give information as to the patency of the tube, presence of exudate in the tube or small amounts of serous fluid in the hypotympanum, and changes in the tension of the drum membrane which cannot be determined by any other method.

The importance of examination of the mouth, throat, nose and accessory sinuses cannot be too strongly emphasized. After preliminary inspection of the nose to determine the condition of the membrane and the amount and character of secretion present, some solution, as 1 per cent cocain or ephedrin, should be employed to shrink the mucous membrane to enable the examiner to obtain a better view of the nasal chambers and note any pathology that may be present. Further information in this area may be obtained by using the postnasal mirror or the nasopharyngoscope to determine the presence of enlargement of the posterior turbinal tips and their relation to the pharyngeal ostium of the Eustachian tube. Adenoids, nasopharyngeal discharge, adhesive bands in the fossa of Rosenmüller, tumor masses and drainage from the posterior group of sinuses may also be discovered by this method. In the mouth and oropharynx attention is focused chiefly on the teeth and tonsils. So frequently apical abscesses, without apparent symptoms, are present, it would seem that X-ray examination of all teeth should be a routine procedure in cases of deafness where the etiological factor is in question. The tonsils are examined for evidence of past or present infection. If the tonsils have been removed there may be an hypertrophy of the lingual tonsil or of lymphoid tissue in the pharynx that may be the seat of infection, and may be directly responsible for the deafness.

The tests for hearing almost universally employed, by laymen as well as physicians, are the spoken and whispered voice and the watch. In using the spoken voice one must not only standardize his voice but the conditions under which the test is made must be approximately the same for each examination. The examining room should be as quiet and free from extraneous sounds as is possible. The patient, blindfolded or with eyes closed, is placed at the farther

side of the room with the listening ear turned to the examiner and the other closed tightly with the moistened finger tip. Whatever the source of sound, it should be brought from a distance toward the patient, otherwise the memory of a sound may cause the individual to think he hears when he really does not. In using the voice one must take into account sound values; vowels are heard farther and with greater distinctness than consonants, and certain vowels, *a, e, i,* are heard farther than others, *o, u;* also certain consonant sounds, as *s, sh,* may be heard two or three times as far as *f* or *th.* And, further, the tones of the different letters are distributed rather widely over the musical scale, *l, m, n,* being in the lower half, while the sibilants are in the upper. It is also wise not to confine oneself to the use of words or numbers alone since with repeated examinations the patients may learn to associate certain sounds with the corresponding word or number and lead the examiner to erroneous conclusions as to gain or loss. By using both words and numbers, consonants and vowels, one can form a fairly definite opinion as to the patient's ability to hear conversational speech.

The watch is not an accurate instrument for testing because no two watches have the same pitch and because the sound produced is not a pure tone. However, if the same watch is used at all times and one knows just how far it can be heard by a normal ear, it does give helpful information.

Tuning forks are the instruments most generally used to determine the state of hearing. Since most individuals are concerned with their ability to hear normal conversation, and since the range of the human voice is from about 200 d.v. to 3,000 d.v., for practical purposes it is unnecessary to use either very high or very low forks, a set of 32, 64, 256, 512 and 4,096 d.v. being sufficient. The method of exciting the fork is important and should be the same at all times in order to obtain uniform results. For the Weber and Rinné tests, an unweighted C_1 fork, which is excited by means of a pleximeter attached to the fork and released by a spring, thus insuring the same impact at all times, has proven very satisfactory. For the low and high tones we have used a 50 d.v. and 4,096 d.v., striking the fork against the knee by letting it fall through an arc of 90 degrees. A number of tests with the forks have been devised but for practical purposes the Weber, Schwabach, Rinné, Gelle and Stenger are the ones most essential. For the technique and interpretation of these tests the reader is referred to any standard text of otology and also to Sonnenschein's[18] excellent description of the various findings with the Rinné.

For testing the higher notes the Galton whistle or monochord is used. The Galton whistle has an upper limit of about 2,000 d.v., but with it one can test by air conduction only. Many investigators consider the monochord very reliable and preferable to the whistle; it has the added advantage that the high tones can be tested by both air and bone conduction.

Within recent years the audiometer has come into fairly general use. With it one gets accurate measurements in sensation-units. The results obtained with this instrument follow rather closely those obtained with the methods above described. The greatest variation I have encountered is in comparison with the Galton whistle findings. Shambaugh and Holderman[20] reached the conclusion that the audiometer cannot be used as a means for determining the hearing in each ear separately, but we have found that, except in cases of total unilateral deafness, the audiometer records lead to the same conclusions as do the records of other functional tests.

Gottlieb[21] calls attention to the contraction of the visual fields as a diagnostic sign in cases of progressive deafness.

When the otologist has exhausted his methods of diagnosis, the internist and serologist must be called upon, for frequently it is only by their co-operation that a correct diagnosis can be established. Complete examination of the blood, including Wassermann test, hemoglobin, cytology and chemistry, is indicated in every obscure case. Diabetes, gout, leukemia and other constitutional disorders having a direct influence on deafness are found only by such thorough methods.

PROPHYLAXIS.

The prophylaxis of deafness or the conservation of hearing is only a method of applied preventive medicine in which the otologist is most actively engaged. The problem resolves itself into the early recognition and correction of conditions which predispose to auditory disease.

Acute congestion of the Eustachian tube producing no symptoms other than a sensation of fullness in the ear, if not recognized and treated may be the forerunner of repeated similar or more severe attacks, which eventually lead to one or more of the lesions of the middle ear, or even to suppurative labyrinthitis and total deafness. But it is not enough to treat only the local congestion; one must search for and remove if possible the pathological condition responsible for the congestion.

In children, the most potent cause of tubal inflammation is adenoids. So constant is this factor that the presence of congestion

or retraction of the membrana tympani in children is regarded as one of the most reliable signs of lymphoid hypertrophy in the nasopharynx. If the adenoid mass is of sufficient size to interfere materially with nasal respiration, the drum membrane is usually retracted to a degree rarely seen in adults. With such findings, removal of the adenoid mass is definitely indicated.

In older subjects, preventive methods must be directed toward the correction of those conditions that are conducive to repeated inflammations of the nasopharynx and to the removal of foci of infection. Local applications to nose and nasopharynx may be all that is necessary in a small number of cases, but where there are repeated attacks of Eustachian congestion, acute colds and so on, one must consider seriously the removal of tonsils, correction of nasal deformities, and investigation of the sinuses and general physical condition.

Since the common cold is so frequently the exciting cause of middle ear disease, one naturally feels that if this cause were eliminated deafness would be considerably decreased. As yet we know very little regarding the etiology and prophylaxis of this common disease. Vaccine therapy as a preventive has many advocates, as well as many opponents. Autogenous vaccine would theoretically be the ideal preparation but, unfortunately, the results from its use have not been encouraging. Likewise, the benefits derived from stock vaccine have not been all that is desired. But in spite of many failures and disappointments, the use of either stock or autogenous vaccine is definitely indicated as a prophylactic measure until some more effective method is discovered. In the office of Dr. Coates we have used both types of vaccine, with good results in many cases. The immunity acquired is not permanent, reinoculation being necessary in from 12 to 24 months.

Diphtheria toxin-antitoxin and scarlet fever antitoxin are decreasing in no small number the cases of deafness resulting from these diseases and are among the most active prophylactic measures we have at our command.

Further prophylactic measures lie in educating the laity to the fact that deafness is largely a preventable disease, and in the frequent examination of school children. Statistics show that there are more than 3,000,000 children of school age in the United States suffering from more or less severe degrees of deafness. The large number of children brought together for eight or nine months each year offers an excellent opportunity to institute some means of conserving the hearing of a large mass of future citizens.

TREATMENT.

The treatment of deafness is extremely unsatisfactory except in a small group of cases where the etiologic factor can be definitely determined and completely eradicated, and in those cases in whom treatment is begun before too great damage has been done to the auditory mechanism.

Deafness resulting from acute inflammatory reactions in the middle ear is usually mild in type and of short duration provided proper treatment is given, and since acute tubotympanic lesions are always due to nasopharyngeal irritations, primary treatment should be directed at the irritative focus. Our treatment in these cases is as follows: The nasal mucous membranes are shrunk with weak solution of cocain or ephedrin, and with a cotton-tipped applicator some of the same solution is applied to the pharyngeal tube mouth. The action of the solution extends along the length of the tube to the tympanum, shrinking the congested tissue and re-establishing the ingress and egress of air. A mild antiseptic solution, neosilvol, silvol or argyrol, is then applied, preferably by spray, to the entire mucous surface of the nose and nasopharynx. Small doses of atropin or belladonna internally are of value in decreasing secretions and preventing congestion. Applications to the nose and pharynx are repeated daily until the marked congestion subsides, and then at less frequent intervals until symptoms disappear, after which gentle inflation of the tube is practiced where indicated. If such a regime is followed practically all acute tubotympanic inflammations will subside, leaving no injury to the auditory mechanism.

Cases not seen until serous or purulent exudate is present in the tympanum with bulging or perforation of the drum membrane and decided deafness are given the same treatment with the addition of particular attention to the tympanic lesion. If the membrana tympani is not perforated or drainage through an existing perforation is not free, the membrane is incised freely. Gentle irrigation with warm solution of boric acid or sodium bicarbonate, or suction by means of the apparatus devised by Dr. S. MacCuen Smith, is indicated in cases where drainage is thick or tenacious. The use, in the external canal, of antiseptics such as mercurochrome, acriviolet and formalin, is probably of very little value in the acute cases unless there is large enough window in the drum membrane to permit the solution entering the tympanum.

In the more advanced cases of conductive deafness and in cases of progressive deafness with involvement of the conductive system, the only hope of relief is in proper attention to the tube and to pathologic processes in the nasopharynx that exercise influence on

the tube. The following method of treatment has been satisfactory in the treatment of cases where tubal narrowing has resulted from a chronic inflammatory process or from repeated acute inflammations: Nasal congestion is relieved by spraying with some mild shrinking solution and weak cocain applied to the pharyngeal tube mouth; the Eustachian catheter is inserted in the usual manner. Air for inflation is heated by passing through a small electric heater attached to the electric circuit, the distal end of this heater being fitted with a chamber containing menthol, camphor, iodin and talcum, and having a tip which fits snugly into the mouth of the Eustachian catheter. When the heated air passes through the chamber, vaporization takes place and the tube and tympanum are medicated throughout. By interrupting the ingress of air at frequent intervals, over-inflation of the tympanum is prevented and a vibratory effect is obtained. After removal of the catheter the tube mouth is treated with silver nitrate solution, 2 to 5 per cent, or some other weakly astringent solution. This treatment is repeated twice or three times weekly at first, the interval being gradually increased, depending upon results. In markedly hypotensed or atrophic drums inflation is contraindicated except perhaps at infrequent intervals, and then very gentle, as a means of preventing adhesions between the membrana tympani and promontory.

When inflation and astringents fail to restore the functional activity of the tube or when there is a definite constriction, some method of dilatation must be used. The applicators, sounds and bougies devised by Yankauer are very satisfactory for this purpose. The applicator, wound with long-fibred cotton for 2 inches and adjusted properly in its handle, is saturated with shrinking solution and inserted through the catheter into the tube and allowed to remain for 20 minutes to one hour. Bougies and sounds are indicated when the applicator cannot be advanced to or beyond the tubal isthmus or where there is no improvement after use of the shrinking solution. The technique is the same as in passing the applicator, but great care must be taken not to use too much force or too large a bougie. The bougie may be left in position for five to 20 minutes and the treatment repeated weekly or biweekly. For the treatment of strictures that cannot be passed without too much force with the usual bougie, Duel[22] uses a gold bougie, which is made the negative pole of a ganvanic current and passes through an insulated catheter.

The value of auditory massage is a disputed subject. There are same cases where some passive motion other than that obtained by inflation seems most definitely indicated. There are a number of fairly satisfactory instruments for massage and if used properly in selected cases they are doubtless of some value.

With marked relaxation of the drum membrane, improvement in hearing is sometimes obtained by efforts to tighten the membrane by repeated applications of liquid collodion or cantharides collodion. The latter must be used with caution because of its decided irritating qualities.

It is evident that the treatments outlined cannot effect permanent results so long as the source of irritation remains, and that in inner ear deafness, due to focal infection, no improvement can be expected unless the focus of infection be removed. Adenoid masses, infecting or obstructing, should be removed. Adhesive bands in the fossa of Rosenmüller should be divided by fulguration or with the finger. Faucial and lingual tonsils and regrowths of lymphoid tissue when definitely infected require removal. Nasal obstructions, turbinal hypertrophies, paranasal sinus disease, carious teeth, require whatever treatment is necessary to eliminate their effect on the ear.

That polypoid enlargement of the posterior tip of the inferior turbinate is an important factor in the production of deafness because of the relation to the tube mouth was emphasized by Sluder.[10] Hypertrophied lateral pharyngeal bands and islands of lymphoid hyperplasia in the pharynx respond in most cases to treatment with astringents. Their treatment by X-ray and its effect on the deafness has been reported by Smyth[23].

Radium has been used with some benefit in treatment of middle ear disease, its value being due supposedly to its loosening effect on old scar tissue and its arrest of inflammatory changes.

In patients with lowered resistance and in certain metabolic disorders, irradiations with the air-cooled mercury vapor quartz lamp or ultraviolet rays may, by their action on the metabolic processes, have a very favorable influence on deafness. Medical diathermy is also of value in certain cases of middle ear deafness.

The treatment of otosclerosis is essentially the treatment of those conditions which I have mentioned as probable causative factors. In early stages, vibratory massage through the external canal may aid in delaying stapedial fixation. Correction of metabolic disturbances offers the most hopeful outlook. Strict hygienic measures and a wise regulation of the patient's mode of living are of great importance. Abstinence from drugs or spirits that are known to produce labyrinthine congestion should be insisted upon. Freedom from mental anxiety, from overwork, and from close confinement to business helps to keep the disease in abeyance. Glandular therapy may be beneficial in selected cases. The experiments of Lewy[24] with calcium fluorid suggest that this preparation may become a practical therapeutic agent.

In occupational deafness, the treatment is limited almost exclusively to a change of occupation. Some protection from the noise may be helpful. Deafness from dust, fumes and gases requires that such causes be eliminated in the places of employment.

Syphilitic deafness is of course treated by active antisyphilitic methods.

Arsenic has long been used, admittedly more or less empirically, in the treatment of deafness. Its action in this disease is not definitely known. The statistics of Prof. Wodak[25], of Prague, relative to results obtained from the use of arsenic in cases of both middle and internal ear deafness, are rather significant.

Finally, there are those unfortunate individuals whose deafness has progressed beyond the stage where treatment of any kind will be beneficial. These patients should be given an honest statement of facts and helped to make a readjustment of their lives. Lip-reading should be encouraged and, when possible, the patient should be directed to some school or individual where the method is scientifically taught. When such procedure is not feasible, some satisfactory mechanical aid should be recommended. These patients should be encouraged to report at intervals to the otologist, not with the idea that their hearing will be improved, but for advice and encouragement and for protection from medical quacks and unscrupulous advertisers.

CONCLUSIONS.

1. By careful study of cases it is possible to classify deafness as perceptive, conductive, progressive or otosclerotic.

2. Such classification is an aid to statistical studies of etiology, clinical signs and symptoms and treatment.

3. The etiological factors most often responsible for deafness of perceptive, conductive or progressive type are the acute infectious diseases, focal infections and nasal and pharyngeal infections and obstructions.

4. Heredity and metabolic disorders appear as the most probable factors in the causation of otosclerosis.

5. Complete history, careful examination of nose, throat and ears, painstaking functional examination, and occasionally studies of blood and metabolism, are essential for correct diagnosis.

6. The results of the audiometer test correspond to those obtained by the usual functional tests.

7. Prophylaxis consists largely in the early recognition and proper treatment of predisposing and exciting causes.

8. Local treatment of the Eustachian tube by inflation with warm medicated vapors, shrinking solutions, bougies and sounds is beneficial in many cases of deafness.

9. Removal of foci of infection, where such foci may be a contributing factor, is indicated in practically every case of deafness.

10. Specific glandular therapy and other necessary treatment of metabolic disturbances seem at present to be the most hopeful means of combating otosclerosis.

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DESTRUCTION OF THE LATERAL SINUS.*

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The following is an interesting case of acute-chronic mastoiditis with complications. Certain unusual features make it worthy of report. That the patient recovered finally is little short of marvelous. At no less than three stages in his illness, he clung to life by a hair's breadth.

HISTORY AND PHYSICAL EXAMINATION.

The patient was a lad of 17 years. His weight was 171 pounds. He had been the athletic type and engaged in spring football practice up to the eve of his illness. His illness, in fact, was occasioned by catching cold after scrimmage.

The patient presented himself for examination on April 6, 1927. He complained of earache, right, for three days prior to this date. He looked sick, though his temperature at this time was only 100°. The right ear was discharging a profuse, foul-smelling pus. Questioning elicited the fact that the ear had been draining more or less continuously for three years. In this period it had never ceased entirely. For the three days of his present illness, the discharge had been notably increased. The right tympanic membrane was practically destroyed by the chronic, purulent process. Granulations were marked over the promontory.

The patient was first kept under observation at home. During this time the temperature was caught on one occasion at 104°; at another occasion at 98.2°; and on several other occasions at slightly above normal. These temperature fluctuations aroused a *suspicion of lateral sinus thrombosis.* What with history and findings, the patient was sent to hospital on April 19, 1927

Preoperative: During the first 48 hours in hospital, the patient developed three chills. The temperature fluctuated between 99° and 104°. On April 22, 1927, a consultation was held. The consultant advised further observation, due to a negative blood culture and a low white blood count. On April 23, the patient had another chill, with a sharp temperature rise from 98.6° to 103.2°. The white count rose to 14,500. On April 24, the patient had another chill, followed by a temperature rise to 104°. Then followed a quick drop

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back to normal. A second blood culture was negative. In the face of the chills and the septic temperature, it was deemed inadvisable to delay operation longer.

Operative: On April 25, just prior to operation at 9:00 a. m., the temperature shot to 105° following another chill. The patient was distinctly losing ground. He looked pasty and very toxic, despite his brief illness of only 10 days. The usual curved incision was made over the right mastoid. The body of the mastoid, including the tip was found to be of the eburnated type. The region of the antrum, however, disclosed a large, practically self-dissected abscess cavity. This was full of dirty, curdled, foul-smelling pus and cholesteatomatous material. The cavity was explored in all directions and found to extend far up and back in the occipitoparietal region. It was necessary to make a secondary incision backward toward the occipital region at right angles to the original wound. The bone was found necrotic for a wide area. Of course, a radical mastoidectomy was performed. Although search was made along its course with field wide open, the lateral sinus was not found. The conclusion reached was that the *lateral sinus had been destroyed by the chronic cholesteatomatous involvement.* The symptoms which had been *thought due to lateral sinus thrombosis were due evidently to this well encapsulated epidural abscess.* The acute process superimposed upon the chronic evidently swung the balance and forced operative intervention. The whole wound was packed with iodoform gauze and left wide open.

The patient's general physical condition during operation could scarcely have been worse. He was extremely cyanotic throughout. Several times he stopped breathing and was stimulated by artificial respiration. Drainage of the epidural abscess cavity was accomplished at high speed. Curiosity prompted inspection of the right jugular. In this respect, however, we were doomed to disappointment. Almost with the incision in the neck, the patient became more cyanotic and showed by pulse and respiration all the evidences of surgical shock. It became necessary to respire him artificially before the open window, administer oxygen and prompt hypodermoclysis. He remained very cyanotic and in precarious condition for many hours.

Post-operative: On April 27, 1927, the first dressing was done. The wound was smeary with pus. No significance was attached to the fact at this time that the temperature ranged as high as 102½°. An interesting feature was the fact that in the face of wide temperature fluctuations during convalescence and numerous complications,

not a single chill occurred. On April 28, the patient complained of pain in his left chest. Examination failed to reveal anything definite. By May 1, his general condition was worse. There was no visible explanation for his continued temperature in the face of the apparently well draining wound. Entirely by accident, while palpating the vicinity of the right mastoid region a small amount of thick, creamy pus was milked upward into the wound from the neck region. Here lay supposedly the explanation for the failure of the temperature to subside. There was no question but what this was *a Bezold-like type of mastoiditis.* For a day or two a rubber drainage tube was inserted from above. This, however, proved inadequate and forced incision in the right neck, on May 7, 1927. A considerable quantity of foul-smelling pus was evacuated. This new wound was drained for several days and then allowed to heal without further event.

It was now expected that the patient would go on to uninterrupted recovery. While he had never been without temperature at any time since his mastoid and neck operations, these two conditions were thought sufficiently explanatory of the fact. When, however, a further rise in temperature to 103° occurred on May 12, 1927, further search for pus was deemed necessary. An internist was called in consultation. On May 14, 1927, the latter reported an area in the left chest suspicious of empyema. The chest was paracentesed and 12 ounces of dirty, foul-smellling pus were evacuated. The patient suffered considerable reaction during the process and all but made his exitus. On May 16, 1927, with X-ray pictures as a guide, a surgeon did a rib resection in the axilla. *A large amount of pus under pressure was released.* A tube was inserted, The patient's condition at operation was precarious. For approximately the next 10 days there seemed a moderate improvement. The patient was bathed frequently in drenching sweats, while the temperature hovered just above normal.

On May 26, 1927, the temperature rose suddenly to 101°. On May 27, it went to 102°; and on May 28, it jumped just short of 103°. The logical explanation seemed to be impoverished drainage from the chest. With this in mind, the surgeon extended the wound in the patient's left side but reported his inability to account for the new rise in temperature on his bare findings. The mastoid wound at this time was healing nicely by granulation and seemed capable of elimination as a factor in the temperature picture.

Inasmuch as the patient's general physical condition each day was steadily growing worse, the surgeon bravely decided on reoperating the left chest. On June 8, 1927, sections of three adjacent ribs were

removed in the post-axillary line. The original pus pocket seemed hardly sufficient cause for the trouble. By great good fortune, a second and larger pocket of pus was found communicating with the first. *This new pocket contained at least a cupful of foul pus.* A large rubber tube was inserted for drainage. The patient barely survived this final surgical procedure.

Convalescence: From this point the patient's recovery proceeded without event. Within 24 hours the temperature had dropped to normal and remained so without a single exception to the end of his hospital stay. The patient was discharged from hospital on June 19, 1927.

Laboratory: The X-ray report on April 22, 1927, showed "right mastoiditis." No hint or suspicion of epidural abscess was given. On May 14, 1927, the X-ray reported "pneumonic infiltration upper right third; considerable fluid left side." Repeated urinalyses failed to show anything of consequence. The WBC on April 20, 1927 was 10,000. The differential showed polys. 86, SM 2, LM 9, trans. 3. On April 23, WBC was 14,500, and on April 30, 29,000. Blood cultures taken on April 22, 24, 26, May 14 and 28 were all reported negative. One blood culture, however, taken on May 3 was reported as showing many staphylococci. This was probably contamination.

Pus from the mastoid wound on April 26 showed Gram positive diplococci and a few short chains of streptococci. On May 3, there were still many Gram positive diplococci and a few small Gram negative bacilli. On June 8, pus from the chest showed streptococci, pneumococci and staphylococci. On June 15, this same pus showed a pure culture of staphylococci.

SUMMARY.

1. This case emphasizes several important points: *a.* The extensive damage possible under chronic mastoiditis; *b.* the seriousness of an acute flareup of such a chronic condition; *c.* the numerous complications, both contiguously and otherwise, that a mastoid breaking down may cause; and *d.* the kindness of nature, which at times permits a sufficient walling off or encapsulation of a pus process in a dangerous locality and seems just waiting for outside surgical assistance. In this instance we were twice favored: first, with the epidural abscess and, second, with the empyema.

2. This case shows the stamina of certain human organisms. The patient in this case was an athlete and had good reserve to draw upon. He went into hospital weighing 171 pounds. He came out of hospital weighing 120 pounds. This represents a net loss of a trifle more

than 50 pounds. When seen, one year later, the patient had recovered entirely his normal weight and strength.

3. The simulation of lateral sinus thrombosis symptoms by an epidural abscess of otitic origin is most intriguing. There was no possible means of arriving at a proper diagnosis in this case except by operative intervention.

4. The complete destruction of the lateral sinus on the right side by the cholesteatomatous process is unique. The author has never seen or heard of a similar case. This destruction must have been accomplished by slow stages. Neither the torcular end nor the jugular end were discovered. Curiosity to investigate patency or collapse of the jugular vein was balked by the patient's near demise at operation. The condition of this vessel will remain in the realm of speculation.

5. The Bezold mastoiditis proved an unwelcome complication. Until this pus was discovered, which had dissected its way into the neck between and beneath the sternomastoid fibres, the patient's failure to improve had been a source of worry.

6. The empyema in the left chest was decidedly a blow. While discovered only on May 4, 1927, it will be remembered that the patient first complained of pain in his left chest on April 28, 1927, exactly three days after his mastoid operation. The process undoubtedly began at this early date and successfully escaped observation. It was not enough to find one large pocket of pus. A second pocket communicating with the first demanded drainage before the patient could rightfully begin his climb up the ladder to recovery.

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CONGENITAL LARYNGEAL STRIDOR.*

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In reviewing the literature on congenital laryngeal stridor one finds some variation in the descriptions of the mechanism producing the stridor, as well as in explanations of its cause. Inasmuch as the term describes the presenting symptom rather than any pathology, this might well be expected. While there has been some tendency to apply this term to a variety of conditions causing stridorous respiration in the newborn, according to Jackson,[1] this should be limited to those cases due to an exaggeration of the infantile type of larynx.

In general the stridor has been noticed at birth, or shortly after. It is inspiratory, although when especially noticeable there may be an expiratory croak as well. This is increased when the child is aroused, and may disappear when quiet. There is no alteration of the normal voice sounds. Cyanosis is rare, although there may be retraction of the thorax and abdomen. Most cases outgrow this condition during the second year, although several fatalities from suffocation have been reported, usually induced by some intercurrent respiratory infection.

Lees,[2] in 1883, was probably the first to report post-mortem findings in a case of congenital laryngeal stridor. These showed the epiglottis curled inwards more than normal and the arytenoepiglottidean folds thin and in close approximation. The rest of the larynx was normal. In 1892, Thomson[3] reported five cases and evinced the belief that the stridor was due to "a central functional disorder consisting in a slight disturbance of the co-ordination which takes place in ordinary breathing between the thoracic muscles on the one hand and those of the larynx on the other". He concluded that it was akin to stammering. Sutherland and Lack,[4] in 1897, reported 18 cases, six of which were examined with the mirror. In these the epiglottis was sharply folded back on itself, the folds approximated and flaccid, flopping to and fro on respiration, and the laryngeal aperture was reduced to a mere slit. Stamm[5] reported a case in which the stridor was constant, continuing during sleep. Cerf[6] made a similar observation, although in his case the stridor diminished in intensity.

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Ballin,[7] in a study of three cases, two by autopsy, found the whole aditus laryngis abnormally narrow, the epiglottis small and beak-shaped, and the arytenoids very close together. Blechmann and Peignaux[8] likewise described the epiglottis as beak-shaped in a post-mortem report. Koplik's[9] case showed the epiglottis laying over the laryngeal aperture with its lateral borders in contact and an approximation of the arytenoepiglottidean folds. Reardon,[10] in 1907, described quite similar findings.

Variot,[11] in his first report in 1898, found the malformation confined to the laryngeal vestibule. In a later post-mortem report in 1917,[12] in a case dying of bronchopneumonia, he found the orifice of the larynx, shape of the epiglottis and the arytenoepiglottidean folds all normal, although all parts were unusually flexible. There was no trace of the posterior cricoarytenoid muscle on the right, while on the left it was somewhat atrophied. From this he concluded that congenital laryngeal stridor might be due to different anomalies of the larynx.

Massei's[13] description of the epiglottis in a report of 11 cases, as imperfectly developed and reduced to a cartilaginous nodule, may well be open to doubt, as it was based upon digital examination alone.

Thomson and Turner,[14] in 1900, reported a study of the larynx in normal infants; from which they concluded that, in the infant, the structures of the laryngeal aperture are soft and collapsible. They reproduced this condition artificially in the cadaveric infant larynx with three different results, constant in each case, as follows: *1.* Sides of the aperture were sucked together. *2.* Epiglottis was drawn down as a lid. *3.* Arytenoids alone met in midline.

Turner,[15] in 1906, again expressed the belief that this condition was due to poor co-ordination of respiration, which produced a narrowing of the laryngeal aperture, with resulting deformity. In the discussion of this paper, Ashby reported post-mortem findings in a case in which the larynx was normal for the age of the infant. He believed the cause to be a "neurosis". Cautley, at the same time, demonstrated a larynx from a case of congenital laryngeal stridor in which there was close approximation of the edges of the epiglottis and the arytenoid cartilages. He expressed the opinion that this condition was due to a congenital malformation, exaggerated by laxity of the tissues. He advanced the argument that the persistence of the stridor during sleep was against the theory of a neurosis. Kelly reported a case in which he found the stridor to be due to the vibration of a flap of loose mucous membrane on the summit of each arytenoid. It is interesting to note that Hasslinger,[16] last year, reported

three cases in which there was an edematous ridge in the mucous membrane of one, or both arytenoepiglottidean folds, which on respiration obstructed the lumen, causing stridor. In two cases these ridges were removed, resulting in immediate relief of symptoms.

In 1922, Thomas[17] reported two cases which came to autopsy, in which the findings in the larynx were normal, while both showed intracranial hemorrhages, and one hydrocephalus. From these, together with three other cases with histories and symptoms indicating cranial injuries at birth, he concludes that congenital laryngeal stridor might be of central origin without laryngeal deformity.

In Putzig's[18] case, autopsy showed a small larynx with a narrow aperture and the cricoid cartilage projecting inwards, causing a defi-

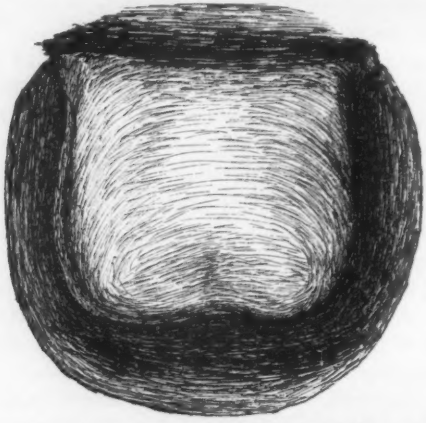

Fig. 1. Direct laryngoscopy. Position of epiglottis and beginning of inspiration.

nite stenosis. The autopsy in Vonderweidt's[19] case showed the epiglottis folded over on its vertical axis, and the arytenoepiglottidean folds very flaccid and laying close to each other. The entire larynx was reported as being only two-thirds the size of a normal one for that age. McKenzie and Williamson[20] reported a case in which the stridor was loudest during sleep. Autopsy showed a flaccid condition of the aperture, with approximation of the folds so as to make with the epiglottis a "cruciform introitus laryngis". Careful examination of the brain and cord showed no lesion of any kind. Ougrelidze[21] reported a case in which there was a definite hypertrophy of the entire right side. No laryngeal examination was reported, but the author was of the opinion that there was an exaggerated development of the right side of the larynx, with overlapping of the folds.

Iglauer[22] reported a very interesting case in which amputation of the epiglottis was performed. There had been several attacks of cyanosis and it had been necessary to resort to artificial respiration. Amputation relieved the asphyxia and the stridor disappeared in 12 days.

While there is some variation in these different case reports as to the exact behavior of the laryngeal structures, it seems to be pretty generally conceded that, in the main, there is simply an exaggeration of the normal infantile larynx, differing from normal only in degree. Even the backward motion of the epiglottis is sometimes seen in normal infants and there is often an approximation of the arytenoepiglottidean folds, so that the aperture of the larynx may be con-

Fig. 2. Right margin of epiglottis beginning to fold under. Early stage of inspiration.

siderably narrowed. The stridor is probably due to the vibrations of the flabby epiglottis and the unsupported arytenoepiglottidean folds being sucked inward during respiration.

There seems to be two theories as to the cause of this condition. There is the central theory, as sponsored by Thomson and Turner,[14] which maintains that this is due to ill co-ordinated spasmodic action of the respiratory muscles, analogous to stammering, and that any laryngeal deformity in these cases is a result of the consequent sucking in of the soft structures.

The other theory is that it is primarily a congenital malformation of the larynx. Sutherland and Lack[4] are perhaps the leading advocates of this idea. They maintain that there is no evidence of any

central lesion and no need of such an hypothesis. The respiratory movements apart from the local changes in the larynx are exactly the same as in normal infants. In reply to the assertion that sometimes a similar condition occurs temporarily in children during anesthesia, probably due to inco-ordination, they say that in this case the noise is made by spasm of the vocal cords rather than the laryngeal aperture, as in the stridor cases.

St. Clair Thomson[23] says that it remains to be settled whether the narrowing of the laryngeal orifice is primary or secondary. Watson Williams[24] considers that both factors enter into the question; that respiratory inco-ordination, occurring in an infant with this peculiar malformation, results in stridor, which is increased by the falling in of the flabby tissues.

Fig. 3. Slightly later stage of inspiration.

In a case recently under my own observation the epiglottis seemed to be the essential factor in the production of the stridor and its movements during respiration were different from any that I have been able to find described.

RESUME OF CASE REPORT.

Baby W., age 2 months. *Family History:* Not significant. *Present History:* Difficulty in breathing since birth. Noisy inspiration. Worse when disturbed. Worst position, upright with head bent forward. Easier when on side. Chokes when feeding. Not on breast—bottle-fed.

Examination: Fairly well developed and nourished baby. Marked inspiratory stridor, with short expiratory croak on forcible respira-

tion. No cyanosis. Voice sound, normal. Heart and lungs, normal (Dr. J. O. Piper). Pharynx, normal.

Fluoroscopy: Both lungs function normally. *X-ray:* Thymic shadow not enlarged. No compression of trachea. Lungs, heart, mediastinum and diaphragm normal.

Direct Examination: No anesthesia. Jackson speculum. Flabby, omega-shaped epiglottis almost horizontal over glottis. Right margin somewhat lower than left. On inspiration right margin begins to roll downward and inward, followed by whole epiglottis, which is sucked down, and then, on expiration is expelled with a croak. This twisting motion is constant. Retracting epiglottis exposes normal-appearing

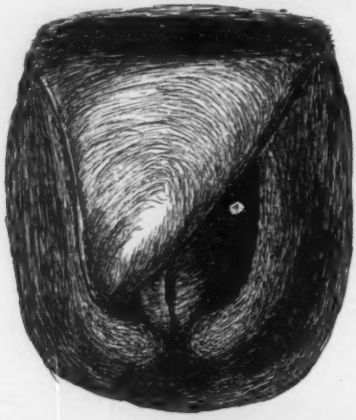

Fig. 4. Right margin of epiglottis considerably rolled under. Late stage of inspiration. Too much of the glottis is shown in this drawing. However, it serves for purposes of orientation.

glottis, although somewhat narrowed, with folds approximated more than normal. This relieves stridor.

The shape of the epiglottis in this case seemed to differ from that usually reported. It was rather wider and not folded back into cylindrical form when above the glottis. On inspiration it began to roll on itself to the right with a sort of twist, so that when sucked down into the glottis the left side was always uppermost.

This peculiar movement of the epiglottis was constant during considerable careful observation. On gentle inspiration the epiglottis was not sucked down into the glottis but the right margin continued to roll inward and downward. Retracting the epiglottis with the tip of the speculum exposed normal-appearing vocal cords and relieved the stridor temporarily. Thinking that perhaps the width and rigidity

of the tip of the speculum might prop the folds apart and prevent their vibrating, the epiglottis was gently retracted by means of a light forceps, trying not to exert any pressure on the folds. This also caused the stridor to disappear.

The mother was given advice as to the general care, feeding, etc., in an endeavor to get the child on as rational regime as possible, and cautioned about the possible necessity for operative interference. She was requested to bring the child back for observation at frequent intervals. Unfortunately, this has not been done. The parents were people of little intelligence and not co-operative in the least. However, I have been able to keep track of the case indirectly through

Fig. 5. End of inspiration and beginning of expiration.

physicians in the locality in which they live. The child has gradually improved during the past year-and-a-half. It has had a circumcision and a tonsil and adenoid operation under ether in what seems ill-advised efforts to correct this condition. Credit for the improvement has been given to these procedures rather than to the natural course of development.

Mosher[25] mentions the asymmetrical larynx with an omega-shaped epiglottis closing over one arytenoid, rather than the whole glottis. Possibly this case might be the forerunner of just such a larynx.

In this case, as in Iglauer's, the epiglottis seemed to be the primary factor in the production of the stridor. Of course the freely movable larynx of the normal infant may sometimes cause the epiglottis to be drawn downward, but here it was sucked completely into the larynx and then expelled with a croak. And when retracted, the

stridor was absent. It is interesting to note that microscopic examination of the amputated epiglottis in Iglauer's case did not disclose any abnormality. In the cases reported by Hasslinger examination of the specimens removed showed simply spongy, edematous connective tissue with few cells and covered with pavement epithelium.

Against the normal laryngeal findings reported by Ashby and by Thomas are the observations of many others, that abnormal conditions were noted in the larynx. It is probably true, as Variot says, that this condition may be due to different anomalies of the larynx. The different laryngeal pictures reported and the variations in the results of the experiments of Thomson and Turner would seem to point to some underlying reason in the structures themselves. Could there be any abnormality in the ligamentous attachments of the epiglottis to the thyroid cartilage, or to the hyoid bone, which might be

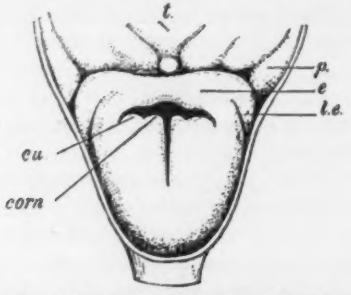

Fig. 6. Entrance to larynx in a 40 to 42-day human embryo (from Kallius): t, tuberculum impar; p, pharyngoepiglottic fold; e, epiglottic fold; l e, lateral part of epiglottis; cu, cuneiform tubercle; corn, cornicular tubercle.

a factor? Could there be any difference in the amount, resiliency or consistency of the cartilage in the epiglottis and upper rim of the larynx causing sufficient flaccidity to produce stridor?

It is generally conceded that the epiglottis and the upper rim of the larynx are the essential factors in, at least, the immediate causation of the stridor. There are a few points concerning these structures which would seem to be of possible interest.

1. The epiglottis and upper rim of the larynx are of different histology than the rest of the larynx. *2.* These structures have a different embryological origin than the rest of the larynx. *3.* These structures are found only in mammals and are subject to considerable variation.

The epiglottis, Wrisberg's cartilages and the apices of the arytenoids are made up of yellow, elastic cartilage.[26] The cartilages of

Santorini are of white fibrocartilage. The rest of the larynx is composed of hyaline cartilage, although occasionally, according to Piersol,[27] elastic cartilage is found in the median part of the thyroid cartilage. Calcification, due to the deposits of lime salts, takes place much earlier in hyaline than in elastic or white fibrocartilage, and while this is not of importance in a consideration of the infant larynx, it points to a fundamental difference in consistency. Yellow, elastic cartilage is pliable, in contrast to the comparative brittleness of hyaline, due to the presence of elastic fibres. Therefore, the epiglottis and upper rim of the larynx, being composed of elastic cartilage, is more pliable and inclined more to flaccidity.

The epiglottis[28] and upper rim of the larynx is developed from the furcula, at about the end of the fifth week of fetal life. This is a horseshoe-shaped ridge separating the pharyngeal aperture of the larynx from the developing tongue. It bounds the laryngeal aperture in front and laterally, and represents the ventral parts of the third visceral arch. This furcula later differentiates into a median elevation which forms the epiglottis, and into two arytenoid ridges, each of which later presents two small elevations, the cornicular and cuneiform tubercules. These form, respectively the cartilages of Santorini and of Wrisberg. The lateral portions of the furcula produce the arytenoepiglottidean folds. Until the fifth month of fetal life the epiglottis lies behind the palate and within the nasopharynx. Incidentally, this is the normal position in the adult in certain mammals. The thyroid cartilage is formed in two lateral portions from masses of mesenchyme, and represents the cartilages of the fourth and fifth branchial arches. Here, then, we see an embryological difference between the upper portion of the larynx and the rest of the organ.

While the glottis[29] is common to the lower animals, commencing with the amphibia, the epiglottis is only found in mammals, although what is supposed to represent the beginnings of the epiglottis is seen in reptiles. This is simply a fold of mucous membrane in front of the glottis. The epiglottis, together with the upper rim of the larynx, is subject to considerable variation in form in the different mammals, possibly due to environmental or habitual requirements. In the cetacea, for instance, the epiglottis and the arytenoids are projected upward toward the posterior choanae of the nose behind the soft palate, forming a long tube with a split at the end. This enables them to inspire air through the nose, while at the same time taking in water containing their food supply through the mouth. This elongated portion is that developed from the furcula. In the marsupials[30]

the epiglottis and upper rim of the larynx is likewise elongated and firmly embraced by the soft palate, so that in the nursing young, the milk may be pumped down from the glands of the mother without interference with respiration. In the dog the cuneiform cartilages are large and crescent-shaped, with the false cords extending from them to the thyroid; while in the ox the cuneiform cartilages are absent. So we see the epiglottis and upper rim of the larynx is a later acquisition in the development of species and is subject to considerable variation in different mammals.

Could we not assume, then, that these same structures in the human infant, of a different histological character, predisposing to flaccidity; and of different embryological origin; a later and more highly specialized development, might, of themselves, be subject to sufficient variation, if not malformation, to produce this condition of congenital laryngeal stridor? The theory that the stridor is caused by a lack of synchronization of the diaphragmatic and intercostal muscles with those of the larynx is appealing, but rather difficult to accept. If these changes noted in the structures are secondary to respiratory inco-ordination, why should the stridor so often be noted immediately after birth. One would expect that some time would be required to produce this flaccidity, and respiration does not occur until birth. Also, it is difficult to see how this theory could account for the part obviously played by the epiglottis in the case reported by Iglauer, as well as my own. As shown by Mosher, the epiglottis normally covers the laryngeal aperture in swallowing, diverting ingested material into the pyriform sinuses, but does not close it. The theory of incoordination means closing, or falling together of the laryngeal aperture with inspiration, causing stridor. The epiglottis, if short and flabby, might be sucked inward, but this would be secondary to the laryngeal rim in causing the stridor. In Iglauer's case, however, removal; and in my case, retraction of the epiglottis relieved the stridor. So it would seem that there must be some local condition, some variation, or congenital malformation, in the structures themselves, of this portion of the larynx, which is the essential factor, in at least, a majority of the cases classed as congenital laryngeal stridor.

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Professional Building.

THE RELIEF OF NASAL OBSTRUCTION IN CHILDREN WITHOUT SUBSEQUENT DEFORMITY.

Dr. William Wesley Carter, New York.

This subject has been much discussed, but there still remains a considerable diversity of opinion as to the best method of procedure. My experience in dealing with nasal deformities, extending over a period of many years, enables me to treat the subject from a basic, clinical point of view, and I trust that my observations may be of some value in helping to decide what attitude should be assumed when a child is presented for the relief of a marked nasal obstruction due to a deflected septum. The temptation to regard the obstructed nose as a mechanical problem, to be solved simply by operation is a strong one, especially as we now know that this may be easily and quickly done. If we yield to this temptation, however, when dealing with children, and do not save and safeguard structures essential to the development and support of the nasal bridge, I am now able to state with reasonable certainty, in the light of knowledge gained by personal, clinical experience, that in due time abnormal development of the nose will become apparent, and, in all probability nasal obstruction will again supervene, owing to the collapse of the soft structures of the nose. These results do not immediately follow the operation; they may not be noted for two years or more, for we must remember that we are dealing with a developmental change and not one resulting from the sudden removal of a mechanical support; the latter would be observable at the time of operation, or certainly within a few days, after swelling had subsided.

Those writers who come out boldly and state unequivocally that the structure of a child's nose is not impaired by an extensive submucous operation certainly draw their conclusions from the condition they find shortly after the operation, and I mean by this within two years at least following the removal of the septum. Apparently they recognize no difference between mechanical and developmental forces in the maintenance of the nasal structure. They miss entirely the point I emphasize and that I have tried for so long to impress upon operators in this field, namely, that in relieving the obstruction in the child's nose, due to a deflected septum, our first thought must be for the preservation of the structures essential to the development

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of a symmetrical organ, and this connot be effected if there is extensive destruction of the nasal septum.

Now, what shall we do in the case of a child whose nose is almost completely obstructed by a deflected septum? It is perfectly plain that to wait until the age of 17 or 18 years is out of the question, for not only will the child's health and hearing be impaired, but a nasal deformity will result anyway, for a badly deflected septum does not perform its normal function, that of raising the bridge of the nose, as it exerts no force in a vertical direction. In its crumpled condition, it bulges laterally, causing the nose to become broad, flat and irregular in shape, with lack of uniformity in the size and shape of the nostrils. This condition is augmented by the negative intranasal pressure, due to the child's efforts to breathe through the nose.

We must keep clearly in mind those portions of the septum that are chiefly concerned in preserving the nasal arch. The upper edge of the quadrangular cartilage is important as a segment of the arch, while the anterior, vertical portion supports the nasal tip. In operating, care must be taken not to dislodge the upper edge of the septum from its position between the two lateral cartilages, for here it constitutes the keystone of the nasal arch and its removal or dislocation will result in a flat nose.

Operation: Every case presents specific problems of its own and to give in detail a method that would be applicable in every case would be impossible, but if the general principles are observed, the variations in the technique, necessary in any particular case, should be obvious to the operator.

Assuming that the patient is a child and that there is a marked deviation of the cartilaginous septum to the left, causing complete obstruction on this side.

The usual vertical incision, down to the cartilage, is made at the mucocutaneous junction on the left side; this incision reaches from the vestibule well down upon the floor of the nose. The mucous membrane is then elevated well back as far as the deviation extends. A narrow strip of cartilage is then removed horizontally from the crest of the deviation throughout its entire length. A parallel incision through the cartilage above and below is then made. In making all of these incisions great care must be taken not to perforate the mucous membrane on the opposite, concave side, which has not been elevated from the cartilage.

We now have the cartilaginous septum, and it may be the vertical plate of the ethmoid as well, reduced to two ribbon-like segments extending from before backwards and attached to the perichondrium on the concave side.

The narrow, longitudinal strip of cartilage having been removed, as has been mentioned, from the crest of the deflection, the upper and lower segments of the septum can now be pushed into the median line without overlapping, and can be held in a vertical position by means of my gold-wire splits, which are fashioned at the time to suit the case and are adjusted in each nasal cavity in such a manner as to hold the septum in a vertical position.

The nasal cavities are cleansed daily with an alkalin solution. The splints are removed in one week. The advantage of the splints over a packing of vaseline gauze is that the nasal breathing is not interfered with and cleansing is more easily effected.

The importance of the anterior portion of the cartilaginous septum as a support to the nasal tip entitles it to special consideration, for if this is destroyed there is certain to be a depression of the tip, a very objectionable deformity, and one that can be corrected only by the transplantation of cartilage.

In order to get a badly deflected anterior edge of the septal cartilage into the median line, it is often necessary, not only to elevate the mucoperichondrium from it entirely, but it may be necessary to sever it near its attachment to the nasal spine and to dissect its upper extremity from its false position beneath the nasal tip. After this is done, if there is difficulty in keeping the cartilage in the midline, I take a reef in the mucocutaneous tissue by removing an elliptical segment, the long axis of which is at right angles to the bulge, and then bringing the cut edges together with fine sutures; this pushes the segments into position and holds them there.

Extreme care must be taken during the healing process to insure protection to the nose and immobility of the parts, as the delicately poised segments of the septum are easily displaced. In most instances it is best to encase the nose in one of my tin-lined copper splints for at least a week.

The results that I have secured by this method are very satisfactory. The normal growth of the nose, so far as I know, has not been interfered with in any of my patients. I attribute this success to the fact that the cartilage segments retain their viability, as the perichondrium is left intact and attached on one side of each segment. The septum, having been restored to a vertical position, gives normal developmental support to the nasal bridge.

CONCLUSION.

1. In relieving nasal obstruction in children, due to deflection of the septum, it is necessary to preserve those portions of the septum essential to development and support of the nose.

2. Untoward results, due to destruction of the septum, do not appear immediately, but only after a sufficient time has elapsed to show the result of interference with the developmental forces.

3. Deformities resulting from this interference can be corrected only by the transplantation of bone and cartilage.

2 East 54th Street.

DIPHTHERIA — DISEASE OF ANTIQUITY — DIPHTHERIA AND TONSILLECTOMY.*

DR. FRANCIS W. WHITE, New York.

The disease under discussion is no new-fangled one. It successfully resisted the inroads of science for nigh unto 2,000 years. A few decades ago, a morbidity in round figures of 10,000 (exact figure, 10,776), such as we had last year, would have meant a death rate of approximately 6,000 if those affected were very young children. However, today it is only 600 (exact figure, 642) for all ages and will be progressively less. The last winter, four cases in particular were brought to my attention at the Manhattan Eye, Ear and Throat Hospital, on Dr. McCullagh's Clinic.

Case 1: A child, age 8 years. Parent stated the patient could not speak plainly, nor swallow; fluids particularly returning through the nose. This occurred after a tonsillectomy 12 days previously. Examination revealed a paralysis of the soft palate, accumulated mucus in the laryngopharynx, paresis of the external recti, diplopia, practically normal accommodation, an unsteady gait, dragging of the feet, and pain in the muscles of the legs; Wassermann and Kahn, negative. An exhaustive and searching case history was gone into and developed the following: The tonsillectomy was performed 20 days *after* the child had been "cured" of diphtheria. Two consultants had dis-

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agreed on a diphtheria diagnosis, but a culture proved positive and antitoxin was given later. Some days before the operation a change in the voice had been noticed, the regurgitation was thought to have been caused by an upset stomach, the eye condition was unnoticed and the other symptoms just simply went with the picture. Strychnin sulphate gr. 1/150 t.i.d. was administered; recovery.

Case 2: Child operated upon for tonsils and adenoids. Several hours after admission, while on the table ready to be operated upon, the surgeon, suspicious of a spot or two on the tonsils, returned the patient promptly to the ward, had a smear made and received a positive diphtheria report in a few minutes. Antitoxin was administered to all patients in the ward. This case has no connection with the next, as the patients were in the hospital at totally different times.

Case 3: A parent came to the hospital and complained that his child had been infected with diphtheria in the institution. The period of incubation of a few hours to a few days being explained to him, it was decided that other sources could be at fault.

Case 4: A case of laryngeal paralysis following repeated intubations. This was a case of traumatic paralysis.

We may pass rapidly over the various forms of diphtheria, namely: Conjunctival—very resistant to treatment—carrier form; otitic; nasal (most potent carrier form); nasopharyngeal; pharyngeal-acute-chronic (carrier); laryngeal (croup) tracheal-tracheo-bronchial; septic or toxic.

Diphtheria and croup at one time were considered separate entities. Today, we know croup may be caused by diphtheria (about 50 per cent), coryza, influenza, measles, and please do not forget the symptoms caused by a foreign body in the upper respiratory tract or an enlarged thymus gland. Fortunately, the X-ray is trustworthy in many instances in the two latter conditions, thus ruling out croup.

In croup during the third stage, the condition is complicated by a pounding heart, circulatory weakness, failing pulse, marked *restlessness* and cyanosis. To offset these or to ameliorate the symptoms, intubation should be done. In the first and second stages, however, suction should be used, followed by adrenalin chlorid locally in the larynx to relieve the respiratory embarrassment. Pneumonias occur chiefly in this form of diphtheria.

Complications of intubation—immediate: *a.* Failure to get the tube into the larynx—note breath sounds. *b.* Making a false passage. *c.* Aspiration of the tube into the trachea—it may even fall into a bronchus—X-ray. *d.* Coughing the tube half-way up, causing

a valve action against the soft palate. *e*. Coughing the tube up into the nasopharynx.

Septic or toxic diphtheria: The membrane is spread over a very wide area and contains both diphtheria bacilli and streptococci. As the name implies, it is an overwhelming infection and demands vigorous treatment. The organ of predilection for greatest damage is the heart. This is a myocardial change and not an endocardial. If a heart murmur is heard it may be an old endocardial change or hemic. Death in the first two weeks in this form of diphtheria is practically always due to a myocardial degeneration. If the patient survives the first two weeks he is then liable to be affected by some form of paralysis, of which there are two forms:

1. Local. This is the more common and may appaer as: *a*. Palatine — nasal voice and regurgitation of fluids through the nose. *b*. Pharyngeal, loss of cough reflex, cannot swallow or expectorate. *c*. Ocular—always *combined* with *palatine*—never alone. *d*. Facial, rarely.

2. General. There is weakness of the muscles of the neck, back and extremities, also the intercostals and the diaphragm. The reflexes are at first hyperactive, then hypoactive, and finally lost entirely. The patient lies immobile and has about as much *muscular* resistance as a *large* piece of *rope*.

Parasthesia—as tingling of fingers and toes, accumulations of bronchial and tracheal secretions may occur, also atelectasis, hypostatic or bronchopneumonia may supervene. Always before administering antitoxin be sure the patient has no tendency to attacks of eczema, hay fever, asthma, urticaria, any form of allergy or unfavorable reactions to biologic injections.

To recapitulate: Case 1 illustrates the toxic effects of diphtheria, local and general paralysis. Case 2. The intimation of a possible complication by operation upon an incipient tonsillar diphtheria. Case 3. The possible legal complication of diphtheria occurring shortly after a tonsillectomy. Case 4. Traumatic laryngeal paralysis due to intubation.

1010 Brook Avenue.

LUDWIG'S ANGINA; CASE REPORT.

Dr. E. G. Gill and Dr. W. R. Whitman, Roanoke, Va.

Ludwig's angina is characterized by an acute spreading infiltration of the soft tissues starting in the floor of the mouth and submaxillary region, which binds all the structures into a hard, board-like mass. The[1] space into which the infection is localized has for its floor the mylohyoid muscle, its lateral walls, the bodies of the mandible, its posterior wall, the muscles which unite to form the base of the tongue and the deep part of the submaxillary gland; for its roof, the tongue and the mucosa covering the floor of the mouth.

Etiology: The[2] most frequent starting point of the infection is from a suppurating submaxillary lymph node or a collection of pus in the floor of the mouth; the general opinion, that this form of infection is usually due to streptococcus.

Pathology: The process is essentially a cellulitis. The fact that pus is not found in many cases on incision does not prove that there is phlegmonous swelling without pus formation.

Symptomatology[3]*:* The onset is marked by difficulty in talking and swallowing, pain in the floor of the mouth and salivation. Elevation of the tongue, with redness and edema of the mucous membrane over the involved area is characteristic. The swelling has usually a board-like hardness. Dyspnea from edema of the glottis may prove alarming and necessitate a tracheotomy. The temperature is not high in most cases, ranging from 99° to 103° F. The conditions from which it is to be distinguished are quinsy and retropharyngeal abscess.

Prognosis: Mortality is high, especially if treatment is not instituted early. Various authors have placed it at about 50 per cent. Death is usually due to edema of the larynx or bronchopneumonia.

Treatment: Incise[3] promptly through the swelling in the submaxillary region, using local anesthesia. Thomas states that the finger should be passed upward into the wound until only mucous membrane intervenes between it and the mouth. Tracheotomy is of life-saving value when there is edema of the glottis.

Case Report: Patient female, age 32 years, was admitted to the Gill Memorial Eye, Ear and Throat Hospital, April 4, 1929. *Chief Complaint:* Severe pain under left jaw and throat, unable to swal-

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low; voice was muffled; saliva was dripping constantly from the mouth.

Past History: Patient states that the left side of her neck began swelling 10 days prior to admission to hospital. Three days after the swelling began, she had a wisdom tooth extracted. The swelling and pain steadily increased.

Physical Examination: Heart, lungs and kidneys, normal. Nose and accessory sinuses, negative. The mouth could be only partially opened. The tongue was swollen and pushed upwards and to the right. The mucous membrane in the floor of the mouth was red and swollen. The tissues in the submaxillary region were swollen and of board-like hardness. The temperature on admission was 101.6°. Leukocyte count, 9,200.

Treatment: Ice packs were applied to swollen parts constantly. Morphia sufficient to keep patient comfortable was administered. Twenty-four hours after admission, patient's condition became much worse. The swelling in the floor of the mouth spread rapidly and forced the tongue out of the mouth. Respiration was rapidly becoming embarrassed. Dr. W. R. Whitman saw the patient in consultation, and an immediate operation, which consisted of a wide incision through the swelling in the left submaxillary region, was performed by him. Blunt dissection uncovered an abscess cavity near the floor of the mouth; about one ounce of pus was evacuated. The dissection was continued up to the mucous membrane in the floor of the mouth; wound was packed with rubber drain and iodoform gauze, and the dressings were kept moist with warm boric acid solution. The alarming symptoms, especially the dyspnea, were promptly relieved, and the patient made a complete and uneventful recovery.

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Gill Memorial Eye, Ear and Throat Hospital.

OPERATIVE HOOK-UP FOR AIR PUMP.

Dr. J. B. H. Waring, Cincinnati.

In the earlier days of the electrically-driven, so-called "tankless", compressed air pump, its principal use by nose and throat practitioners was for spraying or nebulizing various solutions into the air passages; and for aspiration of mucus, blood and saliva from the throat in the several surgical procedures in this region. With increasing usage of compressed air and suction in both medical and surgical practice, a need developed for air pumps at once more powerful; capable of precise regulation; and built for heavy duty usage day in and day out over a period of years. The development of this need slowly witnessed decline of the light, flimsy "portable" air pumps; and perfection of heavier, better built, and more efficient pumps, semiportable or more stationary in type. Aside from efficiency of operation, delicacy of control became an essential. The Barraquer suction technic for cataract operation, for example, requires a specially built air pump, capable of very exact control. Bronchoscopy likewise is making more and more use of the suction pump; and a pump for this purpose must likewise be efficient as to power; yet capable of accurate regulation. Suction tonsillectomy requires a powerful air pump, foot-switch controlled, and likewise accurately controlled.

In our study and development of suction tonsillectomy technique over a period of years, we realized that an efficient air pump was of the utmost importance for perfect work; a necessity: but study of most of the air pumps then on the market disclosed the fact that they were woefully weak and inefficient, and poorly adapted to the work. Study of the Robertson type air pump, a development of the late Dr. John Robertson, of Cincinnati, a decade or two ago, disclosed that here was a basis for perfect results; not only for general all-around office usages of compressed air and suction, but especially for suction tonsillectomy work.

The model selected for development is a duplex, which carries with it the same sturdy build and efficiency we found characteristic of all of these models. Mounted on a wooden base, the pump is sufficiently portable for all practical intents and purposes. Where a more elaborate outfit is desired, the same model may be secured in cabinet style. Pressure and suction pumps run on a common shaft

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and actuated by the same powerful motor, but are otherwise independent. The wooden base model takes up but little floor space and may be plaecd on a chair, table or shelf. Accurate gauges are placed on both the pressure and suction lines; and regulation of power provided by appropriately placed needle valves. Compressed air from 0 to 60 pounds is available, and suction up to 25 to 26 inches at sea level. Attached or attachable devices provide for vibratory massage (positive or negative), along with a set of three suction cups, saliva ejector, etc.

In front of the pump proper on a separate polished hardwood base are placed three metal cups, which hold three reservoir bottles

Fig. 1. (Bone drill attachment not shown.)

of a quart capacity each. It is an absolute necessity that any suction pump be protected by some type of reservoir bottle or receptacle in front of the suction inlet of pump, otherwise mucus, blood, saliva or other material will be drawn directly into the pump, and quickly diminish, if not entirely stop its proper functioning. These three bottles may be placed in any order desired. As a rule we place the first two (suction) bottles in the right and middle holding cups, while the third ether vapor bottle occupies the cup on the left. This ether vapor bottle may be supplied with or without an electric warming device.

The middle suction bottle is connected by flexible rubber tubing to the suction intake proper, and this we designate as the operating

line, governed as it is by gauge and control valve. The tonsil suction tube is now connected to the suction inlet of the righthand bottle by about 4 feet of quarter-inch diameter, flexible rubber tubing. In use, this rubber tubing is suspended at a convenient height in front of the pump, from an overhead cable.

The first suction bottle on the right is now connected by another short length of rubber tubing to the air intake of the compressed air side of the pump; this develops a suction as powerful as the suction pump proper, and yet in action does not in any way interfere with the compressed air pump usage. A saliva ejector of the Yankauer or other suitable type is now connected by another 4 feet of flexible rubber tubing to the intake of the middle suction bottle; the tubing likewise being suspended in front of the pump from an overhead cable. Some operators prefer a suction tongue depressor

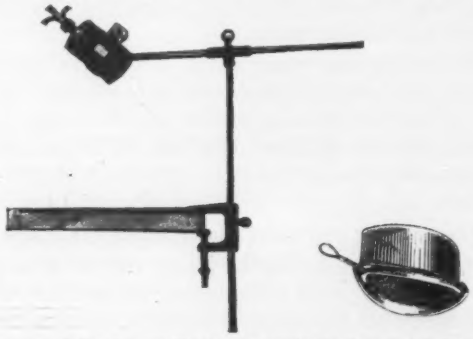

Fig. 2. Improved Yankauer-Waring Anesthetic Cone with Simplex. Dropper arm.

to the saliva ejector, but the hook-up is the same, and merely a matter of preference. If desired, as in case of any trouble with the suction pump promper, this second auxilliary line of suction may be used for operation, but as a rule is reserved for saliva aspiration from the pharynx.

The air pump is foot-switch-controlled, although it is also controlled by a hand-switch. Foot-switch control of the suction pump is a practical necessity for perfect suction tonsillectomy work, and will be found far more efficient in every air pump usage over the hand-switch. We use a plunger type of foot-switch, which rests flat on the floor and starts immediately upon light pressure on the button and continues it in operation only so long as the contact button is pressed down. This gives accurate and instant control of the air pump, which is so powerful that the suction bottles are air-

exhausted almost instantaneously with start of pump; there is no appreciable moment of delay for full suction to develop, as observed with some air pumps.

Suction cleansure of the pharynx is rarely required in a local anesthesia suction tonsillectomy, but is of much value in general anesthesia operation, especially in children. With the independent line saliva aspirator hanging conveniently at hand from the overhead cable, the pharynx may be immediately suction cleaned at any time this is desirable in the course of operation. In operation under general anesthesia, the suction tonsillectomy operator will sometimes find that just about the time his tonsil tube has lifted the tonsil perfeetly from its fossa, and snare play is next in order, the little patient's throat will fill up with mucus or saliva, thus obscuring the perfect view desirable. With the saliva aspirator independently hanging at hand, it is but a second to suction clean the pharynx and proceed with enucleation proper.

While the hook-up may sound a trifle involved as described in the printed word, it is simplicity itself, and very efficient. In the illustration the several air lines are shown coiled in front of the pump, but in actual practice, all three are suspended at convenient height between the pump table and operating table, supported by the common overhead cable, not shown in illustration.

Where the operator desires to employ ether vapor anesthesia, the third bottle on the left, equipped with an anesthesia top instead of suction top, is partly filled with ether (electrically warmed if desired) and connected to the pressure outlet of the compressed air pump, which is previously regulated to deliver whatever degree of pressure deemed desirable. Another length of rubber tubing conveys the ether vapor from bottle to an ether hook or other delivering device at distal end of rubber tubing. This is entirely independent of the suction lines; and in no way interferes with them. At will, ether may be stopped or started by use of a by-pass valve in cap of ether bottle.

In performing an adenoidectomy under general anesthesia, the Yankauer saliva aspirator, or better still, a suction tongue depressor is first carried to the posterior wall of the pharynx with suction on before the adenoid instrument is brought into play. By continuation of suction during the adenoidectomy proper, it is possible to do our adenoidectomy without aspiration of blood and mucus into the lower air passages, or stomach. As blood flows in course of the adenoidectomy, it is instantly aspirated into the suction reservoir bottle, and not only prevents the little patient swallowing blood, but also

obviates the blood and saliva so often seen smeared around and about the little patient's face during an adenoid or tonsil enucleation.

The hook-up above described has been found so entirely efficient and satisfactory, both for suction tonsillectomy work and for the general run of compressed air and suction usage in the office, that it is felt this description may be of value.

Where desired, a flexible cable bone drill may be attached directly to the main shaft of the motor proper, thus affording an efficient motor bone drill for mastoid and all other bone surgery.

7 East McMillan Street.

A COMBINED INSTRUMENT FOR CATHETERIZING THE EUSTACHIAN TUBE AND SINUSES.

DR. LOUIS K. PITMAN, New York.

This is a combined instrument, consisting of a nasopharyngoscope with an adjustable catheter guide. The catheter guide can be so adjusted on the shaft of the nasopharyngoscope, to conform to the anatomy of the nose, for free manipulation for the introduction of a catheter, bougie, etc., into any accessible nasal orifice (sinuses or Eustachian tube).

In Fig. 1, *A* is a nasopharyngoscope having a long range of vision, *T* is a catheter guide, having an outlet *C*, with a flap *D* for guiding the terminal end of the catheter or bougie. The flap is guided by wires *F* (the shaded portion is tunneled, while the unshaded portion between *x*-*x* and also near the flap is free). The degree of deflection of the flap (varying from practically no deflection to marked deflection) is controlled by lever *H*.

The catheter guide *T* is attached to the nasopharyngoscope by a double sleeve *B*. Portion *G* is made to clamp on the nasopharyngoscope in such a manner so as to permit rotation and forward and backward movement of the catheter guide about the nasopharyngoscope. Portion *E* of the sleeve is a tube or a ring, about the shaft of the guide, to permit rotation of the guide on its own axis.

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Manipulation: In Fig. 1, the catheter guide is at the right of the nasopharyngoscope. When in this position, the instrument is accessible for right Eustachian tube catheterization.

To bring the catheter guide in front of the nasopharyngoscope as in Fig. 2, rotate catheter guide on its own axis within ring *E*, for about 90 degrees to the right and then revolve the guide about the nasopharyngoscope by means of sleeve *G*, for about 90 degrees to the left. The instrument is now ready for spehnoidal and frontal sinus catheterization.

To bring the catheter guide to the left of the nasopharyngoscope, as in Fig. 3, rotate again catheter guide on its own axis within ring

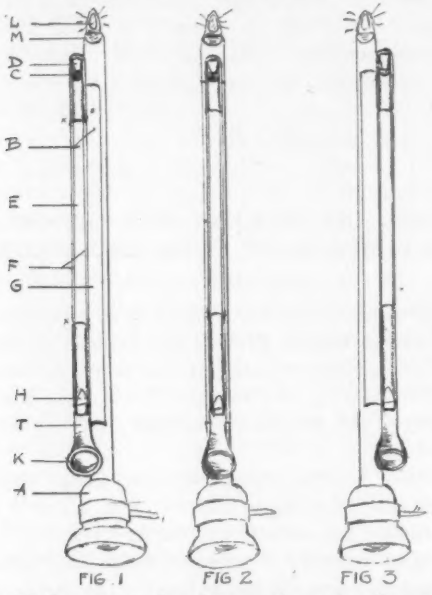

FIG 1 FIG 2 FIG 3

E, for about 90 degrees to the right, and revolve the guide about the nasopharynoscope by means of sleeve G, for about 90 degrees to the left. The instrument is now in position for left Eustachian tube catheterization.

If desired, the catheter guide my be locked, to prevent unnecessary rotation, but is not essential, and therefore a locking device is not described.

The wires guiding the flap may take an inside course, thereby hiding them from view.

910 West End Avenue.

International Digest of Current Otolaryngology.

S. McGibbon, of Edmonton, Canada, submits the following contribution regarding a solution for use with laryngoscopic mirrors. He was in quest of a satisfactory antiseptic which would permit the use of the mirror without drying and warming. There are four prerequisites to a solution for this purpose: *1.* The solution must have a high surface tension; *2.* it must have high adhesive properties; *3.* it must be aqueous so that congealed moisture from the breath will coalesce with it; *4.* it must not evaporate rapidly. McGibbon found that a 1 per cent lysol solution answered all four prerequisites, and, after several months' experience, he has abandoned the procedure of drying and warming mirrors.

Barwich, of Vienna, in the Mschr. Ohrenheilk., October, 1929, reports a new organism as a cause of otitis, meningitis and pneumonia. The organism is the Hemophilum mucosum found by Grekowitz in 1928. It is a Gram negative staff cell which grows easily on blood media and especially easily on Levinthal agar.

Barwich describes a fatal case of pneumonia, bilateral otitis media and meningitis, due to this organism. FINEBERG.

As reported in the British Medical Journal of Oct. 26, 1929, in an article entitled, "Paths of Infection from the Nasal Cavities to the Brain", Professor le Gros Clark applied to the nasal mucous membrane of the rabbit a solution of potassium ferrocyanid and iron ommonium citrate, which solution was found to have reached the surface of the brain within one hour after application. It was found that the solution reached the brain chiefly by way of the perineural sheaths of the olfactory nerves. ROSENBERGER.

Findlay, Macfarlane and Stevenson, in the Archives of Diseases in Childhood, London, October, 1929, discuss tonsillectomy in prevention and treatment of rheumatism. They conclude that a preliminary tonsillectomy does not render an individual less susceptible to chorea but it may possibly have some effect in rheumatic arthritis. A tonsillectomy performed early in the disease renders the heart less likely to be attacked. They make an interesting suggestion that a prophylactic tonsillectomy should be performed before the second attack of tonsillitis, as cardiac complications usually arise between the first and second attack. Carditis following chorea is not influenced by tonsillectomy. They suggest a different strain of the infective agent in these two types of disease. FINEBERG.

Writing in the Ohio State Medical Journal for November, 1929, Lukens, of Toledo, reports one case of fatal hemorrhage following a throat infection.

After reviewing the literature, he summarizes the treatment as falling in three groups:

1. Ligation of common carotid on the affected side.
2. Ligation of the external carotid or the affected vessel.
3. Conservative treatment.

Essayist and discussant recommended ligation of the common carotid and they point out that in children this procedure is but infrequently followed by hemiplegia. A preoperative transfusion is urged. ROSENBERGER.

Nabarro and MacDonald, in the British Medical Journal, Oct. 26, 1929, discuss the bacteriology of tonsils in relation to rheumatism in children. They claim that the streptococci from non-rheumatic tonsils show no difference to those from rheumatic tonsils. From this they conclude that there is no specific streptococcus in rheumatism, but that the condition is probably the result of repeated small doses of toxin. The tonsil is probably one of the foci from which these toxins may be absorbed. FINEBERG.

Dr. John F. Barnhill, of Indianapolis, was chosen as President-elect of the American Academy of Ophthalmology and Oto-Laryngology at its recent meeting in Atlantic City.

In the American Journal of Diseases of Children for November, 1929, Schick and Topper report an interesting study of the immunity to diphtheria conferred on children by tonsillectomy and adenoidectomy.

One hundred children, from 2 to 12 years of age, who had a positive reaction to the Schick test preoperatively, were again tested six months after operation and 82 per cent of them were found to be negative. They conclude that tonsillectomy and adenoidectomy not only eradicate the portal of entry of infection but the operation evidently favors, in some unknown way, the development of an humoral immunity against diphtheria. They make two practical conclusions on the basis of their work: *1.* The recommendation of tonsillectomy and adenoidectomy in place of immunization with toxin-antitoxin for children with diseased tonsils, who are senstive to horse serum; and *2.* the recommendation of testing children who have been tonsillectomized six months or more previously before immunizing with toxin-antitoxin. ROSENBERGER.

Rubin, Epstein and Werner, of Chicago, in the October, 1929, American Journal of Diseases of Children, report the results of blood cultures from 78 patients, following tonsillectomy. All patients had recently suffered infections of the throat. The blood cultures were negative in every instance. FINEBERG.

D'Amato, in the Aug. 29, 1929, Semana Medica, Buenos Aires, presents a report on four cases of chronic gastritis which presented the syndrome of gastric or duodenal ulcer. The diagnosis was made by Roentgen examination. Staphylococcus aureus were recovered from the duodenal conditions. Cultures made from the tonsils showed the same organisms. The author, experimentally in animals, produced lesions on the mucosa of the stomach with these staphylococci. In all four cases tonsillectomy gave excellent results and concludes that in all gastric disturbances where there is a definite chronic tonsillitis, tonsillectomy will very often cure the chronic gastritis.

FINEBERG.

Urbantschitsch, of Vienna, in the August, 1929, issue of the Mschr. Ohrenheilk., presents a very interesting satistical report on operations in acute mastoiditis. Since 1913, he has tabulated 637 opera-

tions in acute cases (within eight weeks of onset). Some of his results are: There were no marked difficulties in relationship to sex; 60 per cent of his operations occurred within the first 20 years of life; 39 per cent of the total, in the first 10 years of life, and 11 per cent in children under 2 years of age; there were no marked differences between the right and left side; infectious diseases were the commonest cases, and of these, scarlet fever was the commonest. Time of operation showed that the greater number were operated on within the first three weeks of onset, gradually diminishing up to the eighth week. Most operations were done during the spring months of the year. He had about 10 per cent of complications (66 cases). Of this figure, sinus thrombosis numbered 40 per cent; sinus thrombosis plus perisinus abscess, 65 per cent; meningitis, 15 per cent. The balance was divided between extradural abscess, labyrinthitis, sinking abscess and zygomatic mastoiditis, about 5 per cent each.

In 637 operations on 533 patients there were 31 deaths, but only 16 of these died from causes related to the ear, which is 3 per cent of the operated cases ending fatally.

He observes that the ear complications (mastoiditis), following measles reached a peak every sixth or seventh year (1914, 1920 and 1927). Another peculiarity observed was that in the past four years he has found 15 cases of sigmoid sinus disease and only 12 cases in the 11 years prior. He explained this by better diagnosis and earlier operations. FINEBERG.

BOOK REVIEWS.

Oreille Interne. Etude Anatomo-Pathologique et Clinique; Technique Microscopique et Expérimentale. By Charles Claoué, Ancien Prosecteur a La Faculté de Médicine de Bordeaux; Assistant a La Clinique du Dr. R. Claoué. With 225 pages and 103 illustrations. Norbert Maloine 27, Rue de l'Ecole-de-Medécine, Paris, 1928.

This book is based mainly on research done by the author. It consists of histological and pathological descriptions of the human internal ear. In place of the usual description of pathology there is a serial section description of the labyrinth with the pathology described separately in each section. Wherever possible the author has correlated the clinical picture with the microscopical findings, thus making it a more attractive book for students.

The volume is divided into three sections: the opening section deals with the embryology, histology and topographical anatomy; the second section has to do with the technique of microscopical sectioning of the temporal bone. The author reports his finding of a new constant landmark which he calls the "bi-commissural line of the internal auditory meatus". The third section describes the author's animal experiments on the technique development of microscopical sectioning of the temporal bone.

The book is profusely illustrated with 103 figures lucidly describing the points under discussion. The majority of the plates are from Claoue's original work and are quite interesting and instructive. M. F.

The Mechanism of the Larynx. By V. E. Negus, M.S. (London), F.R.C.S. (England), Junior Surgeon for Diseases of the Throat and Nose, King's College Hospital, London; Late Arris and Gale Lecturer, Hunterian Professor, and Hunterian Medalist, Royal College of Surgeons of England. With an Introduction by Sir Arthur Keith, F.R.S., London, and Foreword by Max A. Goldstein, M.D., F.A.C.S., St. Louis. 528 pages, with 160 illustrations. St. Louis: The C. V. Mosby Company, 3523 Pine Blvd. 1929. Price $16.50 (cloth).

In this exhaustive volume of over 500 pages, with an interesting Introduction by Sir Arthur Keith, Conservator of the Museum, Royal College of Surgeons, of England, the mechanism of the larynx is described in its evolution throughout the animal kingdom.

Much time and research has been devoted to detail in the study of comparative anatomy in this splendid and elaborate work, and no better tribute can be chronicled than to repeat the observation of Sir Arthur Keith, who states, "There is scarcely a page in this work without a passage in which some new truth is set forth, but with such diffidence, modesty and unvarnished honesty that the unwary may fail to notice their merit."

To find the beginning of the human larynx, the author had to descend to the lowest air-breathing vertebrates. He found that it first appeared as a mechanism for guarding the pulmonary chamber, and this became and remained its chief function.

In his chapter on Speech, which is most interesting and instructive, he states emphatically that one cannot tell by an examination of the larynx whether the owner could speak or not,—"One cannot tell by anatomical means, the larynx of a prima-donna from that of a woman who had the voice of a raven."

The author recognized that "speech is a faculty of the brain and goes so far as to state that a knowledge of the anatomy and motion of the larynx may hamper rather than help a young singer—."

As Sir Arthur Keith mentions in his splendid introduction, "This book contains the results of a decade of untiring inquiry and hard thinking and should be of permanent value for surgeons, physiologists and teachers of surgery."

This extensive research was directed toward the mechanism of the larynx of the whole animal kingdom and solely in regard to the normal mechanism;

therefore, there is no attempt to account for the causes of pathological affections of the human organ.

There are twelve chapters, including Evolution of the Larynx, Modifications for Olfaction, Modifications for Respiration, Modifications for Specialized Mechanism of Respiration, Function of Movements at the Glottis During Respiration, Modifications for Deglutition, Modifications for Regulation of Intra-Thoracic Pressure, Purposive Use of Sound in Relation to the Sense of Hearing, Employment of Sound as a Means of Communication, Mechanism of Phonation, and the Physiological Anatomy of the Human Larynx.

These chapters are subdivided into various groups, each subject being thoroughly considered.

160 splendid illustrations accompany the text, and the bibliography contains 280 references. 15 detailed charts are added as an appendix, describing minutely the anatomical structures from the various forms of animal life to man.

It is an outstanding volume of the subject under consideration, and the author deserves much praise for the time, energy and profound study he has given to this work and the interesting manner in which it is offered.

M. D. L.

Mastoids. By Frederick M. Law, M.D. Volume 1 of Annals of Roentgenology. Second edition. New York: Paul B. Hoeber, Inc., 1929. Price $12.00

The author has had considerable experience both as an otologist and radiologist and discusses his subject from both viewpoints. This lends added value to his interpretation of mastoid X-rays, and his teachings, therefore, are of great use to the practicing otologist. Those cases which present doubtful clinical indications very often do not receive much clarifying information from X-rays because of lack of interpretive signs. It is in just such cases that Dr. Law's book is of most help.

The work is in atlas form. It consists of 33 full-page plates of halftone reproductions of the mastoid.

Among the conditions discussed are acute mastoiditis, with and without cholesteatoma and mastoids with large emissary veins.

Radiological technique, including exposure and development, is fully discussed. M. F.

Kehlkopf-Nasen und Ohrenkrankheiten, Diagnosis and Therapy. By Dr. Richard Kayser and Professor Dr. Walter Klestadt, of Breslau. Fifteenth and sixteenth editions, 216 pages with 152 illustrations. Berlin: Verlag Von S. Karger. Price, Rmks. 9.80.

This latest edition of a standard reference and textbook has been brought up-to-date, with the exception of a description of tonsillectomy, which might just as well have been omitted. An outstanding addition to this edition is the use of the Hasslinger directoscope.

Anatomy, pathology, differential diagnosis and treatment of commoner ear, nose and throat affections are concisely presented and the book should continue to be a useful adjunct to the student's and beginner's library. M. F.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

May 10, 1929.

Chronic Temporosphenoidal Abscess. Read by Dr. E. H. Moyle for Dr. Richard T. Atkins.

Case 1: Male, age 9 years. Admitted to Bellevue Hospital, March 13, 1928. Discharged July 2, 1928.

Present History: Began six months prior to admission with discharge from the right ear, followed by headache and vomiting attacks. The patient was soon confined to the bed because of weakness and loss of weight.

On admission, the patient was unable to walk, due to weakness of the legs. There was projectile vomiting, which had no relation to the taking of food. The temperature was normal.

Physical Examination: Well developed but poorly nourished child, who appeared listless and very helpless, but apparently not in pain. Answered questions slowly; knew only a few things—had forgotten birthday.

Left pupil dilated, reacted only slightly to light; left lid drooped moderately. Both fundi showed marked neuroretinitis with about 4 diopeters elevation of discs. The left retina was very edematous, with many fine hemorrhages about the disc and throughout the fundus. The right fundus showed an area of choroiditis.

Left ear normal. Right canal filled with foul pus. There was sagging of the canal wall. No mastoid tenderness. Whispered voice heard at the meatus. Labyrinth active.

Neurological examination made by Dr. E. D. Friedman. Left memetric facial weakness. Pyramidal tract signs on left side. Spontaneous past-pointing inward with left hand. Percussion tenderness of skull over the right temporosphenoidal region.

Diagnosis: A focal lesion of expanding nature in the right temporal lobe.

Operation: Simple mastoidectomy, right side. Diploetic mastoid. Bone necrotic in the angle between the knee of the sinus and middle fossa, where an epidural abscess was found. From this area free pus was seen to exude through the dura. The dural plate was removed from around this area, exposing a fistulous tract which lead into the brain. A brain needle was inserted and about 3 ounces of pus slowly evacuated. A rubber drain was introduced.

Progress: The patient gradually improved. There was a profuse drainage through the tube.

After a few days the tube was found in the dressing. Lipiodol was introduced into the abscess cavity and an X-ray picture taken, which showed a large cavity.

A Mosher basket drain was now introduced through the enlarged fistulous tract, and was followed by a considerable gush of pus.

The patient then improved rapidly, except for considerable impairment of vision, which was still present on discharge from the hospital.

Case 2: Female, age 19 years. Admitted to Bellevue Hospital, Nov. 30, 1927. Discharged, Feb. 21, 1928.

Past History: Measles and otitis media in early childhood. Discharge from the left ear for six years.

Present History: For several days prior to admission the patient had severe pain in the left ear and left forehead, radiating to the left side of the face and neck.

Physical Examination: Right ear normal. Left ear: foul, purulent discharge; large perforation of drum membrane; middle ear filled with necrotic granulations. Hearing for spoken voice at the meatus. Labyrinth active.

General physical examination negative except for a slight rigidity of the neck. Temperature, 103° F.; pulse, 90; respiration, 20.

Clinical Laboratory: Short chain streptococci in the ear smear. Leukocyte count, 11,200, of which 82 per cent were polymorphonuclears. Spinal fluid was turbid, under increased pressure, with 100 cells per c.m.

Operation: Radical mastoidectomy, left side. Cholesteatoma extending into the zygomatic region. Epidural abscess over the middle ear.

Progress: Temperature dropped to normal after the operation. Less pain. Physical examination negative except for slight rigidity of the neck. Blood culture negative.

Five days after operation, patient complained of pain above the left eye; was fully conscious but did not seem too alert. Ocular fundi somewhat congested. Reflexes still normal. No cerebellar signs. Spinal fluid clear, normal pressure. Leukocyte count, 6,200, of which 86 per cent were polymorphonuclears.

The next day reflexes were increased on the right side; some facial weakness; suggestive temporal anomia. Right ocular fundus shows nasal blur, left fundus normal. Focal lesion in the left temporal lobe.—Dr. E. D. Friedman.

Operation: Exploration of the left temporosphenoidal lobe. A large area of dura was exposed, which was quite tense and partially covered with granulations. A brain needle was introduced for about 2½ c.m. inward and forward, and about 1 ounce of foul-smelling pus evacuated. A Mosher basket drain was inserted and sutured in place.

Progress: Following the operation the patient's general condition markedly improved. The anomia disappeared gradually.

Six weeks following the operation the drain was removed, and after another month's stay in the hospital the patient was discharged. The patient is now apparently entirely well.

Case 3 (acute cerebellar abscess): Female, age 6 years. Admitted to the Harbor Hospital, May 9, 1928. Discharged, Aug. 10, 1928.

Both ears had discharged for 25 days following an acute rhinitis. For several days prior to admission the patient had a moderate elevation of temperature, and severe pain in the head, especially at night. There was a profuse discharge from both ears, sagging of the posterosuperior canal walls, and mastoid tenderness. The temperature on admission was 102.2° F.; pulse, 116; respiration, 24.

A bilateral simple mastoidectomy was performed. The mastoid processes were pneumatic in character, and there was free pus generally throughout, with considerable softening in the region of the antrums and tips. No dura was exposed.

Following the operation the patient was very noisy, restless, and apparently had severe pain.

The wounds discharged a large quantity of pus for the first few days and the temperature oscillated between 100° and 104° F. The patient continued restless, irritable and slept poorly.

Twelve days after the operation the patient was transfused because of the continued elevation of temperature and anemia. (The hemoglobin was 45 per cent; erythrocytes, 3,450,000; leukocytes, 24,500, of which 81 per cent were polymorphonuclears.)

The following day the patient vomited. There was a slight flattening of the right side of the face, and the knee jerks were slightly hyperactive. Later the facial involvement became quite marked. The temperature was 103° F. Both labyrinths responded normally to the cold caloric test.

During the next week the patient was nauseated but did not vomit. She slept very little, and only with the aid of hypnotics. The facial weakness gradually disappeared. The reflexes were normal. The temperature varied between 98.8° and 100° F.

On the twentieth day following operation, the patient vomited again, and had a slight chill. She complained of severe pain in the head, and the pulse was irregular. There was a slight tremor of the right hand on touching the nose with the forefinger.

The vomiting continued and was projectile in type; the pulse was irregular and somewhat slow.

Later the patient became somnolent; the pupils dilated, and there was a fine horizontal nystagmus to the right. There was a marked ataxia of the right upper and lower extremities, and a loss of abdominal reflexes on the right. The temperature was 98° F., and the pulse rate, 60.

The somnolence rapidly increased into semicoma, and the patient was taken to the operating room.

Under general anesthesia, the plate over the cerebellum was removed in front of the lateral sinus, exposing the dura, which was under marked tension. A brain needle was passed into the cerebellum for about 2 c.m. and one-half ounce of thick creamy pus exuded. The patient's general condition immediately improved.

There was not sufficient room to insert a Mosher drain, so a small rubber catheter was used instead.

Culture of the pus from the abscess produced a profuse growth of hemlytic streptococci.

The next day the patient's temperature was 100° F.; pulse, 92; which was regular and of good quality. She was quite bright and able to take some nourishment. Later in the day, however, she had a convulsion, epileptiform in character, lasing for about 1½ minutes. This was followed by several shorter convulsions, and there developed a left external rectus paralysis.

For the next several days the abscess drained profusely; the patient complained of severe pain in the head which necessitated the administration of anodynes. There were no recurrent convulsive seizures.

Gradually the patient's condition improved, aided by the intravenous administration of glucose, and a transfusion of blood. Her appetite returned and she began to take food. She began to sleep naturally.

The external rectus paralysis continued for about two weeks and then gradually disappeared. The ataxia of the right upper and lower extremities also improved.

Except for an occasional gastrointestinal disturbance, which was probably brought about by overeating, the convalescence progressed smoothly.

On Aug. 10, three months after admission, the patient was sent home. The wounds were healed, and the patient's condition was very good except for some ataxia.

After leaving the hospital the patient spent some time in the Adirondacks and later went to Europe.

She is now in good health, and all evidence of ataxia has disappeared.

DISCUSSION.

DR. C. H. SMITH: Were you able to drain the first case from the mastoid wound or did you have to make a trephine opening over the temporosphenoidal lobe and, if so, just how did you do it—with a trephine or with mallet and gouge?

DR. T. J. HARRIS: I would like to know what Dr. Moyle's experience has been with the Mosher drain, and hope he will say a word about that in closing the discussion.

DR. ROBERT LOUGHRAN: I think I saw the second case. The abscess seemed to point more down, and it was almost impossible to get the Mosher drain in. He accomplished wonders with the tube, but with the Mosher drain if he could have had a different angle he could have used it with great satisfaction. I understand he got the drain in but did not feel fully satisfied. There is no question of its value if you get the direct attack.

The child was very ill. I saw it after it got well and the result was really marvelous.

DR. T. J. HARRIS: This has been to us a most interesting case; as Dr. Dwyer has said, it is a most unusual one. We went over the literature of the subject at the time, and it was almost absolutely *nil*. We could get very little light, so far as the ear is concerned, although it is not the first case of caisson disease affecting the ears. As Dr. Dwyer may tell you, the subject will be

presented before one of our national societies next month at Atlantic City. The outstanding feature of the case which baffled us was the discrepancy between the results of the rotation and caloric tests. We repeated our tests, for we could not believe they were correct. We started from the assumption that the two must correspond, and thought there was a fault in the technique; but after we had used the caloric test twice we were convinced they were correct, and we would not subject the patient to any more suffering, which was acute.

Dr. Dwyer's theory as to what has taken place is to my mind a very plausible explanation. At first it was thought we were dealing with an edema, but I think now it is a hemorrhage in the labyrinth. I am not sure that we are altogether right and that it has not extended much further than the labyrinth. One point must be added to the clinical history: when the patient presented himself at the clinic he did not come there for his ear symptoms, but for recurring attacks of partial loss of consciousness and sudden weakness, which was so profound that he would have to sit down and get help, or lean against something. The attacks were a day or two apart, and almost any exertion would bring them on. When I last saw him they were at longer intervals, about a week.

Dr. Dwyer has referred to the audiometer tests. The audiometer did succeed in showing the picture, which we did not understand at all. Had it not been for the audiometer test we would not have felt as encouraged as we did, that there was some hope for the right ear; but that encouraged us to take him into the hospital and give him a thorough course of pilocarpin. I hope you will all look at the records, for it is to us an entirely new picture so far as the etiology is concerned.

DR. E. H. MOYLE, replying to the question about the insertion of the Mosher drain: In these cases the ordinary mastoid operation is performed and the dural plate removed, exposing considerable dura. The abscess is located with the brain needle, and the Mosher drain inserted.

In the case operated on at Harbor Hospital the drain was inserted on the left side.

DR. M. J. GOTTLIEB: I would like Dr. Dwyer to tell us if he thinks the hemorrhages he speaks of are in the various cavities of the cochlea—the aqueductus vestibuli, the aqueductus cochlearis or the aqueductus tympani—or into the auditory nerve or branches thereof. The fact that the condition is bilateral suggests the possibility that it might be a brain stem lesion. The absence of bone conduction would lead one to presume that it was a nerve lesion rather than hemorrhages into the semicircular canals and cavities of the cochlea.

DR. I. HELLER: I would like to ask if the cases that came to autopsy some years ago showed any of these symptoms before death, so as to test the relationship between them and the postmortem findings—and if any examination was made into the labyrinth.

DR. ROBERT LOUGHRAN: It is a very interesting case, for the present generation has not much opportunity of studying such conditions. I talked recently with a young Italian who was connected with a machine shop, and who before he was 18 was a helper in a diving outfit in the harbor and persuaded his foreman to let him go down. He went quite deep and came up suddenly; the only disturbance he had was an intense hemorrhage—he said, from his stomach; it is a question whether a general hemorrhage could be associated with the internal ear. I recall a case of this kind which might have gone on record several years ago; a man who had been treated for an attack of the bends, and brought around to normal pressure gradually, and sent home. When he got home he found that he was deaf. He was sent to the hospital and we went over him and examined both ears, and got into a mass of thick, black muck, and after washing that out we brought his hearing back to normal.

DR. CLARENCE H. SMITH: It occurred to me that it might be that the horizontal canals were affected and that that was why we got no nystagmus on rotation; and that the vertical canals were intact and we got the nystagmus and other reactions on using the caloric test.

Acute Labyrinthitis; Operation; Recovery. Dr. H. B. Blackwell.

It may be that this is not the proper diagnosis and I am presenting this case for an expression of opinion from those present as to what was the matter with this man, and for prognosis. The patient was admitted to the New York Eye and Ear Infirmary on March 21, 1929, with the following history: Twenty years previously, following a fall he had sustained a fracture at the base of the skull, which evidently affected the left petrous pyramid, and since then he has been unable to hear in the left ear. The hearing in the right ear had always been good until the present illness and neither ear had ever discharged. Five weeks before admission, while reading a newspaper just before dinner, he suddenly became aware of an intense headache developing on right side of his head. He cast down his paper and shortly afterwards noticed bright blood appearing in the right canal. There was no earache and he finally ate his dinner and went to bed, slept soundly all night; the next morning on awakening he was surprised to discover that he was totally deaf and very dizzy. The patient had a drunken gait and felt nauseated. I could not observe that the tendency to fall was more one side than the other. Headache developed and became more severe. The same day patient was admitted to a large general hospital, where he remained in bed for the following two weeks. During this time there was some discharge from the right ear. He then became dissatisfied with the hospital and was discharged at his own request and returned home, where he remained for the next three weeks; during all of this time he could not hear the loudest noises and there was no relief from the dizziness and headaches.

Four days before admission to the hospital his symptoms increased in intensity and the right side of his face became paralyzed. On admission patient appeared very sick; temperature, 101.4°. The right side of his face was paralyzed. His headache was so intense, more so on the right side, that he lay on the left side of his head so as to take the weight from the right side, and while there was no stiff neck, he had the facies of a commencing meningitis. He was unable to hear the loudest shout in either ear. Both labyrinths were dead acoustically and statically. Spinal fluid was under great pressure and bloody; 760 cells, 72 per cent lymphs., 28 per cent polymorphonuclear. Blood count showed 7,500 white cells. Aural examination, right, showed a dry perforation in the anterior lower quadrant, no mastoid tenderness. Left side negative.

Two days after admission, I performed a Stacke-Schwartze operation and while there was no pus in the mastoid it was in a very congested condition and bled freely all during the operation. There was some granulations in the antrum and middle ear; the external semicircular canal seemed intact, and there was no solution of the continuity of the labyrinthine wall that was apparent; accordingly, I did not open the labyrinth; the patient was placed back in bed. On the following day the change in his condition was remarkable. For the first time in five weeks his headache had entirely disappeared and his right facial palsy appeared much better, although his deafness and subjective dizziness remained about the same. During the next three weeks we made a number of spinal taps until the spinal fluid became normal. His subjective dizziness has improved, he has no nausea and his facial paralysis disappeared completely five days after the operation. Both his blood and spinal fluid Wassermanns were negative. At present both labyrinths are dead acoustically and statically, and the question is, what was the matter with his labyrinth? Did he have an acute labyrinthitis or a labyrinthin apoplexy, similar in form to Meniere's disease, or a combination of the two? Despite the fact that the cell count of his spinal fluid was 760, I did not feel as though I should open the labyrinth in order to forestall the development of meningitis, although this course of action has been recommended as entirely proper in cases of labyrinthitis with increase of cell count.

DISCUSSION.

Dr. T. J. Harris: The Chairman and the Secretary are to be congratulated on assembling such a group of unusual cases as we have tonight. This is another case which is so unlike anything I have seen in my own experience

that I am not prepared to make a fixed and fast diagnosis. Dr. Blackwell has described the case so clearly and accurately, and his conclusions are so nearly the same as my own that I can merely repeat one or two points of what he has given of the history. I saw the patient two or three days before he did. At that time he was suffering exceedingly from the pain in his head. He gave the history of a sudden onset four or five weeks before and thereafter a slight discharge from the ear, with pain, vertigo and tinnitus. The case had all the earmarks at that time of an acute labyrinthitis; then there was an interval of four or five weeks in which he was able to go around; his headaches had become less, and while he could not hear better, he was going to the clinic as an ambulatory patient. Then came on the attack in which I saw him, and it seemed to be a case that required immediate attention. I did not know what it was, but it seemed as though the cause of the trouble was in the middle ear, and I wish to congratulate Dr. Blackwell on his excellent judgment in doing what he did, and not any more; for I am not sure that if he had done a labyrinth operation the patient would be here tonight.

Perhaps Dr. Blackwell would not insist on the diagnosis of acute labyrinthitis but only that the trouble was in the labyrinth. I shall listen with interest to what Dr. Poe has to say. I am not an enthusiastic believer in the so-called Meniere's disease. I don't know that I have ever seen a true case of Meniere's disease; there are many cases that are so diagnosed, but they are quite unlike the true Meniere's disease; and I should feel that Dr. Blackwell was within safe bounds in regarding this as a case of labyrinthitis, perhaps subacute, but anyway it was the labyrinth that was involved. I shall be glad to hear the neurological view of the pronounced headache. It seemed to me that at that time it was no longer confined to the labyrinth but that he was getting up a meningeal condition, and with the temperature that may suggest itself to Dr. Blackwell. Again, I congratulate him on his successful treatment of this case.

DR. DAVID L. POE: Dr. Blackwell was kind enough to invite me to examine this gentleman to see if I could not advise as to some course of procedure.

These are the notes which I made during the first examination of the patient. To facilitate matters, I shall take the liberty of reading from these notes:

The patient has a normal posture and gait while walking a straight line with his eyes open; but, walking with closed eyes, there is a slight manifest ataxia. There is also a slight deviation to the right. No spontaneous nystagmus. The nasolabial folds are well marked on both sides. The corners of his mouth show a slight drooping, particularly on the right side. There is a slight facial paresis, especially marked in the first branch, so that when he attempts to wrinkle his forehead one side goes up further than the other. Teeth are well shown, whistling is also done well. Adiadochokinesis slight. Finger to nose test good. Response to sweet and salt tests normal. Barany turning tests in all position did not bring about any nystagmus nor any particular evidence of dizziness. There was no past-pointing. No fistula test present. Caloric tests, after the use of 2½ liters of 60° water, did not bring out any nystagmus nor evidences of dizziness. No past-pointing.

The above tests, coupled with the history which the patient gave of a semipurulent discharge from the ear, extreme dizziness, unsteadiness while standing, severe headache, vomiting, temperature, etc., as well as having spent three weeks in one of our large hospitals as a result, then an exacerbation of the symptoms which sent him to the New York Eye and Ear Infirmary to seek relief, led us to the belief that the patient was suffering from a probable latent suppurative labyrinthitis of a possible purulent character. The question uppermost in our minds, under such circumstances, or, might I say, the most vital question under these conditions to be faced is, "What shall be the procedure in this case?" While a great deal pointed to the fact that the patient suffered from a latent purulent suppurative labyrinthitis, yet there were several things present that did not quite fit into the picture which characterizes this disease. A radical mastoid operation was decided upon, as Dr. Blackwell so well stated.

During the operation Dr. Blackwell encountered an enormous amount of bleeding; in fact, the bleeding was the worst he had met with in his 20 years of mastoid surgery. That experience gave us a new thought for consideration.

On again very carefully checking over the patient's history, we elicited the following facts, which were somewhat different from those he gave us previous to the operation. He informed us that 25 years ago he fell off a wagon, as a result of which he suffered a hemorrhage from the left ear, and very shortly thereafter he realized that he was completely deaf on that ear. He does not remember whether he experienced dizziness and nausea. With respect to his present condition, he informs us that while sitting in his home, reading the evening paper, he suddenly experienced excruciating pain in the entire right side of his head. Very shortly thereafter he felt moisture exuding from his ear. That moisture was pure red blood. There was no evidence of it being mixed with pus.

At the New York Eye and Ear Infirmary a lumbar puncture was done. The spinal fluid disclosed the presence of 760 cells per m.m.

We had then the following symptoms for consideration:

Increased temperature, a man who looked very sick, spinal fluid showing 760 cells, severe headaches, information that he was dizzy, slight deviation while walking, complete deafness, and an absolutely dead vestibular apparatus. There was no response to the turning tests nor to the caloric. Almost 3 liters of water of 60° F. was used. There was also a slight facial paresis.

These conditions made us think of several possibilities: 1. Purulent labyrinthitis latent. 2. Hemorrhagic labyrinthitis. 3. Labyrinthitis with meningism. 4. Destruction of labyrinth with an involvement of the central nervous system or base of the brain.

Purulent labyrinthitis with sequester might be ruled out by the fact that while the labyrinth was not opened surgically, the facial paresis cleared up and the temperature remained, with the exception of a very few days, quite normal. The headaches, since the operation, have moderated considerably. The patient has, in addition, attained a large degree of normalcy in his daily intercourse with his fellow men, as well as in appearance.

The case would speak against meningeal involvement, because we cannot at the present time, nor very shortly after the operation, find any meningeal symptoms.

It is difficult to subscribe to a central lesion, because the neurological tests do not disclose any interruption of normal function there. No intracranial nerve is found to be affected; there is no particular disturbance of reflexes, etc.

There remains only the hemorrhagic destruction of the labyrinth for consideration. Taking the history of the patient, the responses or, rather, non-responses to the various labyrinthine tests, showing a dead labyrinth on both sides, the bleeding encountered during the operation, in conjunction with the presense or absence of the other symptoms, leads us to the belief that we are dealing with a clinical case of hemorrhagic labyrinthitis. The transitory presence of the cells in the spinal fluid may be accounted for by the hemorrhage, which was quite diffuse, some of which found its way into the subarachnoid space.

I wish to add my congratulations on the rare judgment Dr. Blackwell used in the choice of operation. The splendid results fully justified the choice.

DR. DWYER: I would like to ask if the blood count and Wassermann were negative.

ANSWER: Yes.

QUERY: Did you find any evidence of fracture of skull at any time?

DR. BLACKWELL: No. The discharge was bloody from the first night, but it was thin for two weeks after.

DR. I. HELLER: Is there anything on record to show the amount of hearing he had before the acute attack?

DR. HARRIS: His hearing was all right with the right ear. The left was dead.

DR. C. H. SMITH: What did the culture from the mastoid show?

DR. BLACKWELL: We did not take a culture from the mastoid.

DR. SMITH: One of the interesting points was the absence of nystagmus and incoordination at the time of the acute symptoms. My theory of the problem is that he lost both of his static labyrinths and the cochlear function on one side at the time of his injury years ago, which could have caused a fracture of the base of the skull; that all he had left was the remaining cochlea, that this went in an explosion of acute suppurative labyrinthitis, which was the condition causing his recent trouble, and that this was accompanied by a protective meningitis. Dr. Blackwell is to be congratulated on bringing him through.

DR. BLACKWELL: What impressed me from the first, was the difficulty in getting a history. He could not hear, and could not see out of one eye; he cannot see very well and cannot hear at all.

The second was, that I felt the man would die when I first saw him, and it was a great surprise to me that he was so much better the next day. At the time of operation his face looked as though it was paralyzed, and the next day it had improved, the headache which had been very pronounced had disappeared entirely and he has had none since. For four weeks prior to that his headaches were very marked.

DR. GOTTLIEB: What was the lesion in the eye?

DR. BLACKWELL: I believe it was a leukoma of the cornea, due to an injury in his childhood, associated with an eccentric pinpoint pupil.

An Unusual Case of Mastoiditis. Dr. John Miller.

This patient, a woman, age 32 years, was first seen by me on March 30, 1929. She came because of neuralgia on the left side of the head, which, while not severe, was annoying enough to lead her to seek medical advice. She gave the following history: About March 1 she had a head cold, followed in 24 hours by a sharp pain in the left ear. Within two or three hours the pain subsided, followed by a serosanguinous discharge, very scanty in amount, stopping completely in less than 24 hours. She paid very little attention to the incident until about a week later, when she felt "neuralgic twinges" on the left side of the head and in the left frontal region. A few days prior to my first examination she had discovered that the hearing on the left side was defective. There had been no discharge from the canal since March 2, nor had there been any pain in the left ear. In addition to the neuralgia, the patient said that she "did not feel right," and was unable to do her work as she ordinarily did, because she became quickly fatigued. Her past history was irrelevant.

On examination, the nose, nasopharynx and throat were negative. There was no evidence of any sinus infection. The right tympanic membrane was negative, but the left one lacked lustre and there was slight redness in Shrapnel's membrane. There was no abnormality of the ear canal, and no definite tenderness over the mastoid. Whisper was heard 2 inches from the ear. The lower tone limit was 128, and the Rinne was positive. By catheterization the middle ear appeared to be clear. Temperature was 99° F. X-ray showed an average-sized, pneumatic mastoid, the cells cloudy, especially in the region between the knee of the sinus and the antrum. There was no distinct evidence of breaking down of cells, but just anterior to the knee there was an area which was suspicious.

During the next 10 days there was practically no change in signs except for a gradually developing slight tenderness over the body of the mastoid just below the antrum region. Temperature ranged between 98.6° and 99.2° F. During this time she lost five pounds in weight. An X-ray, dated April 10, showed no change except that at this time there was no doubt about destruction of bone in the region of the knee of the sinus.

A left simple mastoidectomy was done on April 15. The cortex was very thick. When it was removed there was a gush of pus, apparently from the region of the knee of the sinus, at which point there was a large area of thick granulation tissue replacing the sinus plate. There was not much destruction of cells about the antrum region, but nearly all cells were filled with exudate. All diseased bone was carefully curetted out and the sinus plate which remained was removed until normal sinus wall was seen. The aditus

ad antrum was found to be very small and narrow, and filled with granulation tissue, this apparently sealing off the middle ear from the mastoid antrum.

This case has gone on through an uneventful postoperative period and is now almost entirely healed. On May 1 a whisper could be heard 18 feet, the lower tone limit was normal, the Rinne, positive 35/20.

In 1926, Dr. B. E. Hempstead, of the Mayo Clinic, reviewed the literature and found about 58 cases of definite mastoiditis, in which the middle ear was not affected. Cases of infection followin ginjury to the mastoid process, and cases due to blood-borne infection were included.

Dabney, who is quoted by Hempstead, in defining the condition which he called "idiopathic mastoid abscess," stated that he wished this condition to be considered as "an abscess in the mastoid process of the temporal bone without any *immediately preceding* or accompanying inflammatory involvement of the tympanum." He distinguished idiopathic mastoid abscess from latent suppurative otitis media with or without mastoid involvement by the appearance of the tympanic membrane, which, in the latter is without lustre, full and sometimes markedly bulging. There is deafness and sometimes pain, but no spontaneous discharge of pus. He did not state whether this condition occurred in adults or not, but LeMee and Rendue state that latent otitis media occurs in infants and young children.

Hempstead reported six cases of his own, one of which, Case 3, had had a bloody discharge, length of time of discharge not stated, and pain in the left ear four months before he was seen. When this case was operated a large subperiosteal abscess and perforation of the cortex on the posterior aspect of the tip was found. Hempstead suggests that the original bloody discharge was due to myringitis bullosa. Another case, Case 6, had had a bilateral myringotomy followed by discharge of pus from the ears during a period of four or five days, four months previous to his admission to the clinic. He had both a subperiosteal and perisinus abscess.

In discussing a report of unusual types of mastoiditis in the College of Physicians and Surgeons, in Philadelphia on Dec. 15, 1926, Dr. Geo. M. Coates expressed the opinion that all patients with so-called primary mastoiditis at one time have had middle ear involvement, perhaps mild in character.

I have considered this case as one having some characteristics of so-called primary mastoiditis. The patient was an adult who had no definite purulent discharge from the ear, but only a scanty serosanguinous discharge, for a period of less than 24 hours, nearly a month prior to the first examination. At the time of the examination the tympanic membrane did not appear normal, but its appearance was such that one would not be inclined to regard it with any degree of suspicion, if no previous history relative to that ear had been obtained. The X-ray examinations, showing increasing involvement over a 10-day period, really made the diagnosis and suggested that operation was imperative.

DISCUSSION.

DR. I. HELLER: I think Dr. Loughran has hit the nail on the head, for in many of these cases the infection in the middle ear and mastoid is simultaneous. A number of years ago, I saw a child who had been taken ill that morning and required a paracentesis. I had seen the child a year before and performed a paracentesis at that time, without anesthesia. In the meantime, the child had a pneumonia and the mother did not want to subject it to pain without an anesthetic. The physician gave the child a half-dozen drops of chloroform and it died. That made it a coroner's case and I was present at the autopsy. The child had had no ear symptoms previously; that is, the whole process was less than 12 hours, and yet when the mastoid was opened the blood vessels were all engorged, there was almost as much inflammation in the mastoid as in the middle ear, and there was a little pus in the middle ear. That convinced me that in a large proportion of our cases the infection tackles the mastoid as soon as it does the middle ear.

DR. SMITH: I think the operative findings account for this unusual case; that is, the small plug in the aditus. Heath, of London, makes a point of this

as one of the great causes of mastoiditis. I think what probably happened was that a mild infection started in the middle ear, going back to the mastoid, then this small plug sealed it off and prevented drainage.

DR. MILLER: Someone asked concerning a culture. Unfortunately, the culture which was taken was lost between the operating room and the laboratory. It seemed to me that, with little, if any, breaking down there were fairly good looking cells around the antrum. If there had been more breaking down in that region, I would have thought of streptococcus mucosus, but the cells looked fairly normal. There was no marked breaking down, as in other cases I have seen, where the streptococcus mucosus was present. I suppose this case should not have been called a case of primary mastoiditis but my idea was that most of these delayed cases might be classed as such. I think my notion about that is dictated more or less by reading the opinion of Dr. Coates. In discussing some cases of this type a few years ago, he made the statement that he did not think there was such a thing as primary mastoiditis without some middle ear infection previously.

OFFICERS ELECTED.

Chairman: James Garfield Dwyer.

Secretary: Hugh B. Blackwell.

Advisory Committee:

To serve one year: Edward B. Dench.

To serve two years: Wendell C. Phillips.

To serve three years: Thomas J. Harris.

To serve four years: Richard T. Atkins.

To serve five years: Clarence H. Smith.